SOIL AND GROUNDWATER REMEDIATION GUIDELINES FOR MONOETHANOLAMINE AND DIETHANOLAMINE

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Soil and Groundwater Remediation Guidelines for Monoethanolamine and Diethanolamine
December 2010

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1. INTRODUCTION

The alkanolamine product family consists of ethanol-, isopropanol-, and butanol-substituted amines and includes monoethanolamine (MEA), diethanolamine (DEA), triethanolamine (TEA), and diisopropanolamine (DIPA). The alkanolamines considered in this report include MEA and DEA, and are herein referred to as the "Amines". Common synonyms and trade names for the Amines are summarized in Table 1.

Alkanolamines are bifunctional molecules with both amine and alcohol functional groups. MEA is an aliphatic compound with the formula $NH_2CH_2CH_2OH$ that is produced by reacting one mole of ethylene oxide with one mole of ammonia. DEA is an aliphatic compound with the formula $OHCH_2CH_2NHCH_2CH_2OH$ that is produced by reacting two moles of ethylene oxide with one mole of ammonia. Since their introduction in the late 1920s, the Amines have received widespread use in industrial processes and consumer products (Figure 1). As a result of the widespread application of the Amines, and MEA and DEA in particular, the published literature on the Amines is relatively extensive.

No soil or groundwater remediation guidelines have been published to date for any of the Amines by either Alberta Environment (AENV) or the Canadian Council of Ministers of the Environment (CCME). This document develops proposed soil and groundwater remediation guidelines for MEA and DEA consistent with the Alberta Environment (AENV, 2009a) framework for the management of contaminated sites.

Appendices A and B provide degradation and toxicological data specific to MEA and DEA, respectively, and include tables designated "Table A-1", "Table B-2", etc. Please refer to the appropriate appendices when reference is made to the corresponding table.

2. BACKGROUND INFORMATION

2.1 Chemical and Physical Properties

Table 2 summarizes chemical and physical properties for the Amines. The Amines are miscible in water and have acid dissociation constants (pKa) between approximately 9.5 (MEA) and 9.0 (DEA). Lewis (1992) reported that a 10% (w/v) aqueous solution of MEA or DEA would be strongly basic with a pH around 12. The Amines have low vapour pressures (<1.3 to 53 Pa), Henry's law constants (10^{-6} to 10^{-12}; dimensionless), and partition coefficients (K_{oc} and K_{ow}). The Amines do not partition to lipids as indicated by low bioconcentration factors (BCFs).

2.2 Analytical Methods

The analytical procedures developed for individual alkanolamines can be used for the entire group of chemicals. For this reason, analytical methods for determining MEA, DEA, TEA, and DIPA are reported here. The main analytical methods for determining amines and some of their degradation products include gas chromatography (GC), high performance liquid chromatography (HPLC), and ion chromatography (IC). Analytical methods for the Amines have been developed for water, solids (soil and waste material), and air samples. The following is summarized in large part from Witzany and Fedorak (1996).

2.2.1 Aqueous Samples

Gas Chromatography (GC)

Amine analysis by GC can be conducted with or without derivatization techniques. Many of the recently reported GC methods use direct aqueous injections and/or columns that are amenable to aqueous injections and thus, do not require sample derivatization.

Methods using Derivatization Techniques

Piekos et al. (1975) reported a GC/flame ionization detector (FID) method for the analysis of MEA, DEA, and TEA. The method involved derivatization with N,O-bis(trimethylsilyl)acetamide, separation with a glass column packed with 3% OV-1 coated on 100/200 mesh Diatomite CQ, and detection by FID.

Choy and Meisen (1980) also derivatized with N,O-bis-(trimethylsilyl)acetamide to determine DEA and its degradation products. Because aqueous solutions of up to 90% water were analyzed, and the tolerance for water was only 5%, samples were dried in Erlenmeyer flasks in a sand bath at 80°C under a stream of dry air. The residue was dissolved in dimethylformamide and the solution was then derivatized with N,O-bis(trimethylsilyl)acetamide and analyzed using GC/FID with a column of 8% OV-17 on Chromosorb W HP 80/100 mesh (6 ft x 1/8 in O.D.). Choy and Meisen (1980) suggested this method was better suited to the separation of degradation products than the separation of MEA and DEA.

A sensitive GC/FID technique for detecting ethanolamines and isopropanolamines in air samples has been reported by Langvardt and Melcher (1980). The methodology included sampling, desorption, lyophilization, and derivatization steps. Derivatization by heptafluorobutyryl imidazole in dichloromethane was used. The phase separated dichloromethane layer was analyzed by GC/FID following separation with a 1.7 m x 2 mm I.D. glass column with the packing prepared by coating 1% (w/w) phenyldiethanolamine succinate over a specially deactivated bonded polyglycol 80/100 mesh diatomite support. MEA, DEA, monoisopropanolamine (MIPA), DIPA, TEA, and triisopropanolamine were examined and recovered with yields near 90% at concentrations from 0.1 ppm (v/v) to 12 ppm (v/v) in air (36 L sample).

Methods not using Derivatization Techniques

Direct aqueous injection has been used in Alberta. Samples are injected into a 30 m NUKOL column (0.53 mm I.D.) in a GC equipped with a FID. The detection limits for water and soil samples were 0.005 mg/L and 0.05 mg/kg, respectively. The method used for soil analyses involved extracting a 5 g sample with 5 mL of water.

Shahi et al. (1994) described a GC technique for analyzing aqueous acid gases, alkanolamines (MEA, DEA, methyl-DEA (MDEA), 2-amino-2-methyl-1-propanol), and their degradation products from natural gas sweetening without sample preparation. Separation of the lighter, more quickly eluted components, as well as the alkanolamines and their degradation products required two columns in parallel with a switching mechanism similar to an earlier method (Robbins and Bullin, 1984).

A Tenax GC column (6 ft x 1/8 in) and a Haysep Q packed column (8 ft) were used by Shahi et al. (1994) for gas separation. For samples containing only CO_2, amines and their degradation products, a single Tenax GC column was found to be sufficient for separation.

Dawodu and Meisen (1993) evaluated four different column types for the analysis of fresh and chemically degraded alkanolamines in aqueous solutions using a GC/FID. The Supelcowax 10 (15 m x 0.53 mm I.D., 1.0 µm film thickness) was found to be superior to the Tenax TA packed column (Supelco), the DB-Wax capillary column (Chromatographic Specialities), and the HP-17 capillary column (Hewlett-Packard). Sensitivity of the Supelcowax 10 column was established using an aqueous solution containing nine alkanolamines at concentrations ranging from 0.01 to 0.05 mol/L. The Supelcowax 10 was able to separate MEA, MDEA, and DEA and showed better reproducibility at lower concentrations.

Boneva (1991) reported a procedure for separating MEA, DEA, and TEA in the presence of ethylene glycol without derivatization. The technique involved GC/FID and a 20M Carbowax wide-bore fused-silicia capillary column (25 m x 0.53 mm I.D.). Kennard and Meisen (1983) developed a technique for analyzing chemically-degraded DEA solutions. Direct injections of aqueous samples were performed using a GC equipped with a 6 ft x 1/8 in O.D. stainless-steel column packed with Tenax GC (Alltech) and a FID. Good separation of MEA, DEA, and TEA and degradation products was found with this method and concentrations of 0.5 wt.% were analyzed accurately with this procedure.

At the Shell Calgary Research Centre in Calgary, sludges containing sulfolane and DIPA (and their thermal degradation products) were dissolved in methanol and analyzed using a packed column (6 ft x 1/8 in O.D., containing Poropak PS; 80/100 mesh) in a GC equipped with a thermal conductivity detector (C. Drury, personal communication, 2001).

High Performance Liquid Chromatography (HPLC)

Hayman et al. (1985) developed an HPLC technique for biogenic amines in which DEA and MEA were separated from a mixture of amines. Aqueous solutions of amines were derivatized with dansyl chloride and extracted with ethyl acetate. Analyses were performed using a Varian HPLC system with a LC 5000 solvent delivery system, solvent programmer, and a fluorescence detector. The column used was a reversed-phase Spherisorb C_{18} column (5 µm ODS, 25 cm x 5 mm I.D.) with a guard column (5 cm x 5 mm I.D.)

A method of air sampling, derivatization, and analysis by reverse-phase HPLC was described by Serbin and Birkholz (1995). Sampling was performed by either midget impinger or by pumping air through a silica gel tube. Ethanolamines were desorbed from the sampling matrix using methanol, water, and HCl and the resulting solution was buffered between pH 7.7 and 8.9. Fluorenylmethyl chloroformate was used to derivatize the samples for ease of detection after separation. Derivatized samples were analyzed using a Varian 5000 liquid chromatograph equipped with a Waters Model 420 fluorescence detector and a Supercosil LC-8 column (25 cm x 4.6 mm I.D. 5 μm). MEA and DEA were detectable at 1 μg per silica gel sampling tube. Calibration graphs were linear over a 100-fold concentration range. MEA, DEA, and DIPA were measured in air with detection levels of 0.13, 0.07, and 0.06 ppm, respectively, based on 3L sample volumes.

Ion Chromatography (IC)

Gallagher et al. (1996) developed a new method of analysis using ion chromatography to study the biodegradation of MEA in environmental samples. This method addressed the problem of extraction of MEA from soil. The extraction method used was 100 mM HCl with 1% chloroform to inhibit microbial degradation during extraction in a 1:10 solid to liquid ratio (w/v). The extraction fluid and soil were mixed by wrist action shaker, settled overnight, and a portion of the solution was decanted and centrifuged. The sample was diluted from 1:2 to 1:10 and analyzed using a Dionex 2010 system (IonPac CS14 cation exchange column with an IonPac CG14 guard column) with gradient pumps and a conductivity detector. This system resolved MEA and ammonium. Based on 10 tests with MEA-spiked soil, the extraction efficiency was found to be 93.3% with a range from 86.6% to 98.0%.

Mrklas et al. (2003) describe a technique for the analysis of MEA in environmental groundwater samples. Their method involved using cation exchange chromatography and conductivity detection. Analysis was carried out using a DIONEX 2000i IC equipped with a 25 μL sample loop. The eluent was 6mM methanesulphonic acid at a flow rate of 1 mL/min. The regererant was distilled deionized water at a flow rate of approximately 2 mL/min.

Krol et al. (1992) evaluated methods for cation-exchange separation and ion interaction separation of alkylamines and alkanolamines in complex sample matrices such as wastewaters and scrubber solutions. The HPLC system was configured with a Waters Model 600 solvent delivery system and a Model 431 conductivity detector. The IC-Pak Ethanolamine cation exchanger (50 x 4.6 mm I.D.) was considered appropriate for low ppm analysis of amines in samples containing large amounts of sodium and ammonium. The Waters IC-Pak and Cation M/D cation exchanger (150 x 4.6 mm I.D.) were found to be more appropriate for trace level amine analysis. The method had a detection limit of 0.025 ppm and was linear from 0.025 to 20 ppm.

Other Techniques

Qureshi et al. (1990) demonstrated a rapid and sensitive test to detect μg quantities of aliphatic amines. A Whatman No. 1 filter was impregnated with 2% diphenylcarbazide and a drop of amine solution was placed on the filter. An immediate pink-violet color indicated that aliphatic amines were present in solution. Detection limits for MEA, DEA, and TEA were 1.60 μg, 1.29 μg, and 0.89 μg respectively.

In their biodegradation studies of eight different amines, including MEA and DEA, Emtiazi and Knapp (1994) used a spectrophotometric method of analysis. They found that interfering materials in environmental samples, including river waters, activated sludges and soils, were insignificant in their amine analyses.

Other methods for analyzing DEA and its degradation products include infrared and ultraviolet spectroscopy, and paper and thin-layer chromatography. These methods suffer from various disadvantages including lack of accuracy, specificity, reliability, and simplicity (Shahi et al., 1994). Determination of individual components of ethanolamine mixtures can be performed by chemical methods, although these methods have been found to be nonspecific and, for the most part, inaccurate (Brydia and Persinger, 1967).

2.2.2 Soil Samples

While most laboratories have been able to quantify the alkanolamines effectively in aqueous samples, analysis of soil samples has proved much more challenging. Data compiled by Tindal et al. (2007) indicated that analysis by two commercial laboratories of samples of MEA and DEA spiked into a range of soil matrices resulted in alkanolamine recoveries that were poor (often 5 to 50%) and not repeatable. These analyses were based on aqueous extractions at various pH values. It appears that such extractions are not capable of recovering these alkanolamines quantitatively and reliably from all soil matrices.

Accordingly, Alberta Environment commissioned a study to develop an effective extraction technique for alkanolamines. The method developed involves refluxing the soil sample with

0.01M HCl for 1 hour. Full details are available in Appendix C. Performance testing of the method is reported for 3 soils, 3 spiking levels and 4 alkanolamines, with 5-8 replicates of each sample. Average alkanolamine recovery over all was 97%.

The method presented in Appendix C is recommended for analyzing alkanolamines in Alberta. Alternative methods are acceptable, but must meet or exceed the performance criteria in Appendix C.

2.3 Sources and Emissions

MEA and DEA are used in a wide variety of applications including gas purification, surfactants and detergents, textiles, metalworking fluids, agricultural chemical intermediates, cement grinding aids, and cosmetics (Figure 1; summarized from Knaak et al., 1997). The total worldwide production capacity of amines in 1992 was estimated at 300,000 metric tons, and the U.S. production capacity of amines in 1995 was estimated to be 447,727 metric tons. The following summary of amine production and use has been compiled from Knaak et al. (1997), Davis and Carpenter (1997), and Sorensen et al. (1996, 1998).

Gas Purification. MEA and DEA are used at sour gas plants where their function is to remove acid gases such as CO_2 and H_2S. MEA is one of the most common solvents for treating gas streams with low to medium concentrations of CO_2 and H_2S. DEA is used under conditions of higher acid gas concentrations and in the presence of COS and CS_2.

Surfactants and Detergents. Amines are important intermediates in the production of surfactants because of their dual functional groups. They are used to form amine salts and control pH. MEA acts as a foam stabilizer, corrosion inhibitor, and rinse improver in heavy duty, dry, powdered detergents. DEA is used in liquid laundry and dishwashing detergents.

Textiles. Amines are widely used in the textile industry where they serve as intermediates for producing cationic softening agents, fabric finishes, dye agents, and lubricants. Major uses include ultraviolet light fade inhibitors, antistatic agents, and fiber treatment.

Metalworking. The Amines are reacted with acids to produce inhibitors that prevent metal corrosion by penetrating and oxidizing the outside layer of the metal. In oil-based formulations, they act as emulsifiers by accepting corrosive water-soluble materials.

Cosmetics. Amines are added to shampoos, hair conditioners, and creams where they act as foam improvers and thickeners.

Other Important Uses. The Amines are used to control corrosion in oil-drilling mixtures, in water treatment, and in mixed solvent systems such as ethylene glycol antifreeze. Amines are used as plasticizers in polyurethanes and as intermediates in the manufacture of glues, adhesives, rubber, and herbicides.

2.4 Distribution in the Environment

The Amines may be released to the environment from industrial facilities, disposal of consumer products, agricultural chemicals in which it is used as a dispersing agent, or in urine. Despite their wide-spread use, however, little data have been published on the distribution of amines in the environment. Background concentrations of MEA in surface waters in Japan (<0.0003 mg/L; n=27 samples) and in seawater from the NW Atlantic Ocean (0.0002 mg/L) were reported in Verschueren (1983). In the NW Atlantic Ocean near the Columbus Islands, an air concentration of 0.043 $\mu g/m^3$ MEA was reported in Verschueren (1983). In an abstract, Robins et al. (2002) noted that amines have been detected in soil and surface water near natural gas processing facilities in western Canada, but did not report concentrations.

2.5 Human Exposure

Based on the physical and chemical properties of MEA and DEA, human exposure can occur via soil and water, but is unlikely via the atmosphere, due to the negligible vapour pressure of these compounds (Table 2). Exposure via food and consumer products is possible for MEA and DEA.

No regulatory estimates of the daily human exposure to MEA or DEA were available.

In the absence of supporting information, the human estimated daily intake, the ambient air concentration and background soil concentration are all assumed to be zero in areas isolated from facilities where the Amines are used.

2.6 Existing Criteria, Guidelines and Standards

Very limited information was found concerning guidelines, criteria and standards for the Amines.

Canadian Federal

CCME (1999 and updates) soil quality guidelines have not been developed for MEA or DEA, but have been developed for DIPA. CCME (1999 and updates) water quality guidelines have not been developed for MEA or DEA, but have been developed for DIPA.

Health Canada (2008) has not developed Canadian Drinking Water Guidelines for any of the Amines (MEA, DEA, MDEA, TEA, or DIPA). Health Canada (2004) has not published Tolerable Daily Intakes (TDIs) for any of the amines.

Canadian Provincial

Alberta Environment (AENV, 2009a, as amended) has developed soil and groundwater remediaton guidelines for DIPA. The British Columbia Ministry of the Environment has not developed soil and/or water quality guidelines for MEA or DEA, but has developed water quality guidelines for DIPA. The remaining provinces in Canada have not developed soil and/or water quality guidelines for MEA or DEA

US Federal

The U.S. EPA (2005) does not publish a water quality guideline for any of the Amines (MEA, DEA, MDEA, TEA, or DIPA) protective of aquatic life, or a Maximum Contaminant Level (MCL) for any Amines in drinking water. The Amines are not included in the list of chemicals for which the U.S. EPA publishes Ecological Soil Screening Levels (EcoSSLs).

US State

No criteria, guidelines, or standards were found in a limited search of state information.

Europe

No criteria, guidelines, or standards were found in a limited search of European information.

Global

The World Health Organization (WHO, 2004) does not include the Amines in its "Guidelines for Drinking Water Quality, Third Edition".

Occupational

The American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value-time-weighted average (TLV-TWA) standard is 3 ppm for both MEA and DEA. The STEL is 6 ppm for MEA and 15 ppm for DEA. The TLV for DEA was derived from the no observed adverse effect level (NOAEL) of 20 mg/kg-bw/day from the Smyth et al. (1951) 90 day rat feeding study and a safety factor of 10. The national Institute for Occupational Safety and Health (NIOSH) recommended exposure limit (REL) for both compounds is 3 ppm.

3. ENVIRONMENTAL FATE AND BEHAVIOUR

3.1 Adsorption and Mobility

The Amines are miscible in water and have low K_{oc} values (log -0.223 to -0.308; Table 2), and therefore they would not be expected to sorb significantly to organic carbon in the soil. For uncharged organic compounds, a low K_{oc} value implies mobility in the subsurface. However, the acid dissociation constants (pK_a) values for the Amines (9.68 and 9.01 for MEA and DEA, respectively, Table 2) indicate that they will be largely protonated and would exist as cations within a typical environmental pH range and will tend to sorb to the charged surfaces of clay minerals. Accordingly, the distribution coefficient (K_d) for the Amines will be controlled by the cation exchange capacity (CEC) of the soil. The Amines are expected to be relatively immobile in most soil-water systems in Alberta. However, in sandy soils with low CEC, or in highly saline soils, it may be possible for the Amines to be more mobile.

The Amines act as weak bases in aqueous solution, and thus adding these compounds to a soil water system will tend to increase the pH. It is possible that a large release of MEA or DEA could increase the pH of the soil sufficiently high such that a significant amount of the Amines would be present as the non-protonated form. Under such conditions, it is possible that the initial mobility of the Amines close to the release would be higher than otherwise expected. However, transport of the Amines outside the immediate spill area would bring the amine compound into a zone of more typical environmental pH values where the mobility was once again controlled by soil CEC.

MEA

Soil-water K_d values have been determined experimentally for MEA. Sorensen et al. (1997) conducted batch equilibration tests using an Alberta soil with MEA concentrations of 10, 100, and 1,000 mg/L, and pH values of 6.5, 7.5, and 8.5. The K_d values determined under these conditions ranged from 2.21 to 4.91 (Table 2). For the purposes of guideline development, the conservative (low) end of this range was selected and the value 2.21 was adopted for the K_d of MEA (Table 3).

DEA

Soil-water K_d values have also been determined experimentally for DEA. Sorensen et al. (1998) conducted batch equilibration tests using four soil from Alberta, Louisiana, New Mexico, and North Dakota. DEA concentrations used were 10, 100, 500, and 1,000 mg/L, and pH values of 6.5, 7.5, and 8.5 were tested. Measured K_d values ranged from 1.9 to 6.4 for four soils of varying clay content and CEC at a pH of 7.5 (Table 2). For the purposes of guideline development, the conservative (low) end of this range was selected and the value 1.9 was adopted for the K_d of DEA (Table 3).

Sorensen et al. (1998) also investigated the effect of ionic strength on the K_d of DEA. They showed that the K_d value decreases with increasing solute ionic strength, ranging from 0.001M to 0.1 M K_2SO_4. They also concluded that with increasing ionic strength, DEA mobility increased from immobile (K_d>10) to intermediate mobility (K_d = 0.5-2.0).

Other Amines

The above findings for MEA and DEA appear to be broadly consistent with those of Luther et al. (1998), who showed that DIPA adsorption was a function of CEC and pore water salinity. In a detailed study of DIPA partitioning using clays (montmorillinite and kaolinite; CEC = 81 cmol/kg and 10 cmol/kg, respectively), hummus-rich soil (3.6 wt.% carbon), and site soils (CEC = 3.7 to 24 cmol/kg), Luther et al. (1998) showed that DIPA K_d values ranged from 3-5 L/kg for sandy soils to approximately 40 L/kg for montmorillinite. K_d values for silty clay till soils in southern Alberta ranged from 14-24 L/kg. Two lines of evidence suggested that sorption was a function of CEC. First, sorption coefficients were curvilinear, with the slope decreasing with concentration. Second, sorption decreased with increasing pore water salinity.

3.2 Aqueous-Phase Solubility

The Amines are reported by a number of sources to be miscible in water (Table 2).

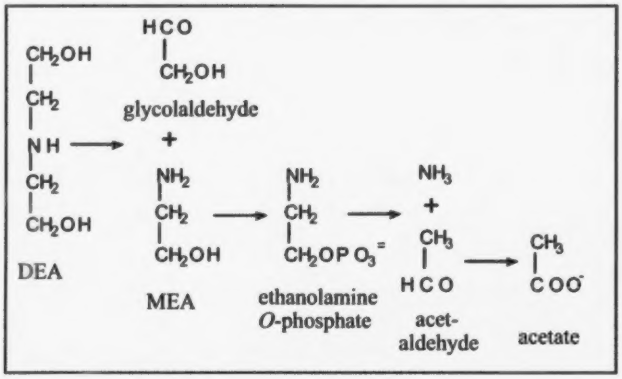
3.3 Leaching and Lateral Movement

Based on the miscible nature of the Amines, it is expected that they will leach from discrete waste sources (e.g., filters in landfills at gas plants). The lateral movement of the Amines will depend on the texture of the aquifer material and the salinity of the pore water. For clay-rich soils, lateral movement would be expected to be limited. Lateral movement could be significant for coarse-grained material, and for salt-impacted aquifers.

3.4 Biodegradation

3.4.1 Degradation Pathways

Williams and Calley (1982) isolated a gram-negative bacterium from a laboratory-scale activated sludge plant treating an effluent containing cutting fluids, that could grow on MEA, DEA, or TEA as its sole carbon and energy source. The degradation pathway proposed for MEA and DEA is illustrated below. TEA was oxidized to triethanolamine-N-oxide, which was subsequently cleaved to DEA and glycolaldehyde. DEA was metabolized to MEA and glycolaldehyde. MEA was activated to ethanolamine O-phosphate, which was subsequently degraded to ammonia and acetaldehyde. The phosphate group was released, allowing it to be used again by the cell.



Ndegwa et al. (2004) proposed a similar pathway for the degradation of MEA, where the ethanol groups split from the MEA were oxidized to CO_2 via acetaldehyde and acetic acid, and the ammonia oxidized to N_2 via nitrite and nitrate.

3.4.2 Inhibition of Biodegradation

Sorensen et al. (1997) demonstrated that an MEA concentration of 1,500 mg/kg in soil increased the lag time prior to biodegradation starting, suggesting possible inhibition of bacterial activity at this level. However, subsequent work by Mrklas et al. (2004) found that degradation of MEA was active at concentrations as high as 31,000 mg/kg.

Gannon et al. (1978) found that DEA inhibited biodegradation at 2,000 mg/L, while Emtiazi and Knapp (1994) found no inhibition or toxicity at 10,500 mg/L (Table B-1).

3.4.3 Degradation Rate

The CCME (1991) protocol for developing water quality guidelines protective of freshwater aquatic life from acute toxicity data requires a determination of the chemical's persistence. In this context, persistent is defined as a half-life greater than 8 weeks in surface water. The AENV (2009a) model for remediation guidelines protective of freshwater aquatic life includes a parameter value for the degradation rate of the chemical in an aquifer. The discussion of amine degradation rates provided below is focussed on i) making a determination of the persistence of these compounds for the purposes noted above, and ii) determining a suitable value for the degradation rate in the AENV (2009a) model.

Data on the degradation rate of the Amines are provided in Tables A-1 and B-1 for MEA and DEA, respectively. Data in these tables are categorized based on whether the studies are potentially relevant to subsurface conditions. Tests conducted under unamended conditions and/or anaerobic conditions are considered potentially relevant to subsurface conditions.

Determination of Persistence

Many datapoints are available in the above-noted tables for studies conducted under amended conditions ("Other Studies" in Tables A-1 and B-1). Most of these studies demonstrated that, with suitable amendments (sewage, bacterial cultures and/or other amendments) MEA and DEA can be significantly degraded within 5 to 20 days. These studies were mostly designed to determine whether these chemicals could be effectively degraded in municipal water treatment facilities. However, they have some relevance to the likely persistence of these compounds in surface water bodies, where oxygen and nutrients are typically available. Based on the significant and relatively rapid degradation indicated in the "Other Studies" sections of Tables A-1 and B-1, both MEA and DEA are considered non-persistent in surface water.

Determination of Subsurface Degradation Rate

In contrast to the situation in surface water, degradation rates for many compounds in groundwater are limited by the availability of nutrients and/or electron acceptors. Accordingly, the degradation rates for studies conducted under amended conditions may have little relevance to likely degradation rates in an aquifer. Studies with data from unamended microcosms, or other conditions potentially relevant to groundwater, are discussed below.

MEA

Several studies have been completed that have relevance to estimating the degradation rate of MEA in the subsurface.

Mrklas et al. (2004) investigated the degradation of a mixture of MEA, ethylene glycol and triethylene glycol in slurries of contaminated soil and groundwater collected from a decommissioned sour gas plant (Table A-1). The study was designed with the objective of determining the potential for in-situ degradation of these compounds at the decommissioned sour gas plant. The initial level of MEA in the slurry was approximately 31,000 mg/kg. Aerobic and anaerobic studies were conducted on both biotic and abiotic bioreactors. The concentration of MEA was monitored directly using cation exchange chromatography with suppressed conductivity detection in water mode. Aerobic reactors received an addition of phosphate on day 11 or 64. Aerobic studies indicated that MEA degradation was limited by the availability of phosphate. Based on interpretation of data presented, in the absence of supplemental phosphate, the aerobic half-life (time to reach half the initial concentration) of MEA was approximately 225 days. With supplemental phosphate, the aerobic degradation of MEA was much more rapid, with a half-life of approximately 4 days. Anaerobic reactors were supplemented with phosphate at day 11, but the addition of phosphate to these reactors had no apparent affect on degradation rate. Anaerobic data were interpreted as zero order degradation, with an anaerobic half-life of 275 days.

Sorensen et al. (1997) investigated the biodegradation of MEA in soil at a moisture content of 30% of field capacity under aerobic and anaerobic conditions. The initial concentration of MEA was 500 mg/kg. MEA was analyzed by ion chromatography. Test soils were not amended with nutrients or inoculated with a bacterial culture. Aerobic studies indicated a lag time of approximately 4.5 days followed by degradation at a constant rate (zero order kinetics). The aerobic half-life of MEA was approximately 13.5 days. Anaerobic studies also indicated linear kinetics, however, the lag time was less well defined by the data. The anaerobic half-life of MEA was approximately 80 days.

Ndegwa et al. (2004) investigated the biodegradation of MEA in samples of an Alberta clay-till soil from a gas processing plant located in northwestern Alberta. The soil was contaminated with MEA and degradation by-products. Some test samples were also spiked with additional MEA. Tests were conducted at ambient moisture content. Aerobic and anaerobic studies were conducted at ambient temperature and at 5°C at a range of MEA concentrations. These authors found rapid degradation under all conditions investigated, with a MEA half-life of 2 to 7 days, and total degradation in 8 to 41 days. Degradation rates were slightly faster in anaerobic than aerobic conditions, and slower at 5-10°C than at ambient laboratory conditions.

Gallagher et al. (1996) studied the biodegradation of MEA at a sour gas plant in southern Alberta. Uncontaminated soil samples were used to determine whether aerobic, MEA-degrading populations could be enriched in laboratory cultures under various incubation conditions. MEA was added to the soil at concentrations of 400, 950, and 1,500 mg/kg, and CO_2 measurements were made over a 120-day incubation period. Incubation temperatures were 6°C, 14°C, and 25°C. Reported lag times were 24, 9.5, and 5.3 days, respectively. Gallagher et al. (1996) measured the decrease in MEA concentration in cultures incubated aerobically at 25°C. Uncontaminated soil was used as the inoculum and MEA was added to give an initial concentration of 500 mg/kg. The biodegradation rate was 29 mg/kg-day (corrected by Witzaney and Fedorak 1996) to yield a half-life of approximately 9 days. Gallagher et al. (1996) also studied anaerobic degradation of MEA in an uncontaminated soil that was spiked with MEA. No external terminal electron acceptor or other nutrients were added to the uncontaminated soil, which was incubated in serum bottles at 25°C under a nitrogen atmosphere. No degradation of MEA had occurred after 32 days.

Overall, the most relevant degradation study was considered to be Mrklas et al. (2004), based on the following considerations:

- **Unamended**. The study showed that aerobic MEA degradation can be phosphate limited, and the first 64 days of some tests were conducted without the addition of phosphate or other amendments.
- **Aerobic and Anaerobic**. Data were available for both aerobic and anaerobic conditions.
- **Direct Analysis**. MEA degradation was monitored by direct chemical analysis, rather than an indirect method such as respirometry.
- **Relevant Substrate.** The study was conducted with a slurry of soil and groundwater from a decommissioned sour gas plant in Alberta that had used MEA.
- **Relevant Concentration.** Initial MEA concentrations were relevant to conditions at a decommissioned sour gas plant in Alberta; and,
- **Relevant Moisture Content.** Data from a slurry study is more relevant to aquifer conditions than data from studies on soils at typical soil moisture contents.

The MEA half-life of 275 days interpreted from the Mrklas et al. (2004) anaerobic tests has been selected for use in the calculation of remediation guidelines (Table 3).

DEA

Only one study was available that had relevance to estimating the degradation rate of DEA in the subsurface.

Knapp et al (1996) investigated the anaerobic degradation of DEA under nitrate-reducing conditions. DEA-degrading bacteria were isolated from anaerobic river sediments collected from the River Aire in the town of Leeds, England. Anaerobic microcosms were supplemented with nitrate and phosphate. The initial concentration of DEA was 5 mmol/L (525 mg/L). The DEA concentration reduced to approximately 1.5 mmol/L (158 mg/L) after 40 days, after which, little further degradation was noted. Data interpretation indicated that degradation was limited by the availability of an electron acceptor (nitrate) and followed zero order kinetics. The interpolated time for 50% degradation was 29 days, which was interpreted as the degradation half-life from this test.

The following factors were considered in estimating a subsurface degradation rate for DEA.

- The database of studies for DEA degradation that are relevant to subsurface conditions is very limited.
- The Knapp et al. (1996) study was conducted under anaerobic but amended conditions; thus, may not be conservative for all subsurface situations.

- The half-life for MEA estimated earlier in this section is significantly higher than that estimated for DEA.
- Degradation of DEA is though to proceed via MEA, and thus the degradation of MEA may be a rate-limiting step for DEA degradation.

Considering the above issues, it was decided that the conservative MEA degradation half-life of 275 days would also be used for DEA (Table 3).

3.5 Volatilization

Volatilization of the Amines is low and is not expected to be significant in the environmental behaviour of these chemicals. The vapour pressure and Henry's law constant for MEA are approximately 53 Pa and 10^{-6} (dimensionless), respectively. The vapour pressure and Henry's law constant for DEA are <1.3 Pa and 10^{-12} (dimensionless), respectively (Table 2). In relative terms, DEA will volatilize less than MEA.

3.6 Photolysis

Based on a photochemical reaction with OH^{o}, the half-life of MEA in the atmosphere was calculated to be approximately 27 hrs (Verschueren 1983). Photolysis data for DEA was not identified.

4. BEHAVIOUR AND EFFECTS IN AQUATIC BIOTA

4.1 Freshwater Aquatic Life

Toxicological data for freshwater aquatic life for MEA and DEA are provided in Tables A-2 and B2, respectively. As required by the CCME protocol the studies have undergone classification into Primary, Secondary, and/or Unacceptable categories. Studies classified as Primary or Secondary are discussed for each chemical below.

Primary and Secondary freshwater aquatic life toxicity data for MEA and DEA are illustrated in Figure 2. Data are presented separately for each group of organisms. Chronic data are presented as solid symbols and acute data use hollow symbols.

4.1.1 MEA

Seven studies were available that were classified as Primary or Secondary (Table A-2). These studies are discussed below. Additional studies that were of Unacceptable data quality are also included in Table A-2 for completeness, with the reason for excluding them as Primary or Secondary data sources. These studies are not discussed further.

Bridie et al. (1979). In this study, the authors determined the acute LC_{50} of 87 chemicals including MEA to goldfish (*Carassius auratus*). Values of 170 mg/L and 190 mg/L were obtained for the 96 hour LC_{50} for MEA. These values are broadly consistent with the 96 hour LC_{50} of 105 mg/L for rainbow trout (Vizon, 2006). This duration is considered acute for these species.

Bringmann and Kuhn (1980)

Much of the considerable body of work published by these two authors is available only in sources which are either unpublished, foreign language, or both. However, this English language paper summarizes the methods and results and covers a good portion of their work. This wide ranged study tested the effects of 156 degrading, organic, contaminant chemicals in water, including MEA, on three selected test organisms known to be relatively sensitive to contaminants (*Scenedesmus quadricauda, Entosiphon sulcatum, and Pseudomonas putida*). The durations of the tests were 7 days, 3 days, and 16 hours for *S. quadricauda, E. sulcatum, and P. putida,* respectively. Growth was measured by increased turbidity, which reduced the transmission of monochromatic light with a wavelength of 436 nm, and was evaluated relative to controls. The calculated endpoints were the concentration required to cause a 3% reduction in light transmission relative to controls (the IC_{03}). The lowest endpoint from this study for MEA was the 7 day IC_{03} for the green alga *S. quadricauda, which* was 0.75 mg/L. This endpoint is considered chronic for this species.

de Zwart and Sloof (1987). This study was designed to investigate the toxicity of mixtures of chemicals, but also includes 48 hour LC_{50} values for 3-4 week old clawed toad larvae (*Xenopus laevis*) exposed to 33 single chemicals including MEA. The 48 hour LC_{50} for this species was 220 mg/L. This duration is considered acute for this species.

Geiger et al. (1990). This book is a large compilation of acute toxicity data for the fathead minnow, and is out of print. The fathead minnow LC_{50} for MEA from the U.S. EPA (2010b) ECOTOX database reported in Table A-2 (2,070 mg/L) is less sensitive than the LC_{50} for rainbow trout (Vizon, 2006), therefore the data from Geiger et al. (1990) were not used to develop the MEA guideline. The original source was not reviewed for this data point.

Groth et al. (1993). In this study, the authors determined the acute toxicity of a range of amines including MEA and other chemicals to fertilized zebrafish (*Danio rerio*) eggs. The 96 hour LC_{50} was determined to be 60.3 mmol/L (3,684 mg/L). This duration is considered acute for this species.

Roseth et al. (1996). In this study, the authors determined the acute toxicity of a range of oil industry process chemicals, including MEA, to the growth of the alga (*Isochrysis galbana)*, and to the survival of zebra fish fry. The 96 hour EC_{50} for alga was determined to be 80 mg/L. This duration is considered chronic for this species. In the zebra fish fry test, no effect was found at 5,000 mg/L, the highest concentration tested.

Vizon (2006). This study was commissioned to fill data gaps in the literature such that at least the minimum requirements for developing a CCME interim guideline were met. Vizon (2006) conducted 96 hour static lethality tests using rainbow trout (*Oncorhynchus, mykiss*) and the freshwater amphipod (*Hyalella azteca*), and a 48 hour static lethality test using water flea (*Daphnia magna)*. Environment Canada biological test methods were used throughout (EPS 1/RM/9 for rainbow trout, EPS 1/RM/33 for *Hyalella azteca*, and EPS 1/RM/11 for *Daphnia magna*). All the requirements for Primary data quality were met, including measured chemical concentrations. Results are provided in Table A-2. The lowest acute LC_{50} was 67 mg/L, which was the 48 hour result for *D. magna*. This duration is considered acute for this species.

Overall, these data suggest that alga are the most sensitive group to MEA toxicity, with invertebrates and fish being less sensitive.

4.1.2 DEA

Thirteen studies were available that were classified as Primary or Secondary (Table B-2). These studies are discussed below. Additional studies that were of Unacceptable data quality are also included in Table B-2 for completeness, with the reason for assigning them to this category. These studies are not discussed further.

Turnbull et al. (1954)

This study investigated the acute toxicity of a range of chemicals including DEA to the bluegill (*Lepomis macrochirus*), with an exposure duration of 1 to 2 days. The LC_{50} from the 2 day test was 1,850 mg/L. This duration is considered acute for this species.

Wallen et al. (1957)

This study investigated the acute toxicity of a range of chemicals including DEA to the western mosquitofish (*Gambusia affinis*), with exposure durations from 1 to 6 days. The LC_{50} from the 6 day test was 560 mg/L. This duration is considered acute for this species.

Bridie et al. (1979). In this study, the authors determined the acute LC_{50} of 87 chemicals including DEA to Goldfish (*Carassius auratus*). The American Public Health Association method number 321 for static tank acute toxicity tests was followed. The 24 hour goldfish LC_{50} for DEA was 800 mg/L. This duration is considered acute for this species.

Bringmann and Kuhn (1980)

This wide ranging study tested the effects of 156 degrading, organic, contaminant chemicals in water, including MEA, on three selected test organisms known to be relatively sensitive to contaminants (*Scenedesmus quadricauda, Entosiphon sulcatum, and Pseudomonas putida*). The durations of the tests were 7 days, 3 days, and 16 hours for *S. quadricauda, E. sulcatum, and P. putida,* respectively. Growth was measured by the increased turbidity, which reduced the transmission of monochromatic light with a wavelength of 436 nm, and was evaluated relative to controls. The lowest endpoint from this study for MEA was the 7 day IC_{03} for the green alga *S. quadricauda,* which was 4.4 mg/L. This duration is considered chronic for this species.

LeBlanc (1980)

This paper reported the results of 48 hour static acute *Daphnia magna* toxicity tests with a wide range of industrial chemicals. Standard U.S. EPA test methodology was used. The 48 hour LC_{50} for *D. magna* was 55 mg/L. This duration is considered acute for this species.

Mayes et al. (1983)

These authors investigated the relative sensitivity of different life-stages of fathead minnows (*Pimephales promelas*) to nine chemicals including DEA. In the case of DEA, little difference in chemical sensitivity was found for the different life-stages, with the lowest result being 1,370 mg/L for the 96 hour LC_{50} for sub-adult fish. This duration is considered acute for this species.

Cowgill et al. (1985)

These authors studied the effect of varying temperature on the 48 hour static acute toxicity of four chemicals, including DEA, to water fleas (*Daphnia magna* and *Ceriodaphnia dubia*). The lowest result from these tests was 29 mg/L for the 48 hour LC_{50} for *C. dubia* at 24.5 °C. This duration is considered acute for this species.

Gersich et al. (1986)

These authors investigated the precision of 48 hour static acute *Daphnia magna* tests with seven chemicals including DEA. Triplicate tests were conducted, and the geometric mean of the three 48 hour LC_{50} values was 116 m/L. This duration is considered acute for this species.

de Zwart and Sloof (1987). This study was designed to investigate the toxicity of mixtures of chemicals, but also includes 48 hour LC_{50} values for 3-4 week old clawed toad larvae (*Xenopus laevis*) exposed to 33 single chemicals including DEA. The 48 hour LC_{50} for this species for DEG was 1,174 mg/L. This duration is considered acute for this species.

Geiger et al. (1990). This book is a large compilation of acute toxicity data for the fathead minnow and is out of print. The fathead minnow LC_{50} for DEA from the U.S. EPA (2010b) ECOTOX database reported in Table B-2 (4,710 mg/L) was not used to develop the DEA guideline. The original source was not reviewed for this data point.

Cowgill and Milazzo (1991)

This detailed study examined the toxic effects of seven chemicals including DEA to water fleas (*Daphnia magna* and *Ceriodaphnia dubia*. Mortality was assessed at 2 and 6 days (*C. dubia*) and 8 days (*D. magna*). Three reproduction endpoints were assessed over three broods for each species: total progeny, number of broods, and mean brood size. The duration of the three brood reproduction tests was 7-10 days for *C. dubia*, and 9-11 days for *D. magna*. The lowest of the mortality endpoints was 19 mg/L for the 6 day LC_{50} for *C. dubia*. The lowest of the reproduction endpoints was 34 mg/L for the EC_{50} for total progeny over three broods with *C. dubia*. The reproduction endpoints in this test are considered chronic.

Warne and Schifko (1999)

This study investigated the toxicity of a range of laundry detergent components to *Ceriodaphnia dubia*. The 48 hour EC_{50} for DEA was 73 mg/L. This duration is considered acute for this species.

Vizon (2006).

This study was commissioned to fill data gaps in the literature such that at least the minimum requirements for developing a CCME interim guideline were met. Vizon (2006) conducted 96 hour static lethality tests using rainbow trout (*Oncorhynchus, mykiss*) and the freshwater amphipod (*Hyalella azteca*), and a 48 hour static lethality test using water flea (*Daphnia magna)*. Environment Canada biological test methods were used throughout (EPS 1/RM/9 for rainbow trout, EPS 1/RM/33 for *Hyalella azteca*, and EPS 1/RM/11 for *Daphnia magna*). All the requirements for Primary data quality were met, including measured chemical concentrations. Results are provided in Table B-2. The lowest acute LC_{50} was 344 mg/L, which was the 96 hour LC_{50} for *H. azteca*. This duration is considered acute for this species.

Overall, these data suggest that alga are the most sensitive group to DEA toxicity, with invertebrates being less sensitive, and fish significantly less sensitive.

4.2 Marine Aquatic Biota

Toxicological data for MEA and DEA for marine aquatic life are provided in Tables A-3 and B-3, respectively. These data are included for completeness, but are not otherwise relevant to this report and are not discussed further.

5. BEHAVIOUR AND EFFECTS IN TERRESTRIAL BIOTA

5.1 Terrestrial Plants

MEA toxicological data found in literature for terrestrial plants have been compiled in Table A-4. Nine papers were identified that reported data on six different plants. However, none of these papers contained toxicological data that linked plant responses to concentrations of MEA in soil; hence, these papers were not relevant to guideline development for MEA. No data were found in the literature on the toxicity of DEA to terrestrial plants.

Accordingly, definitive (14 or 21 day) growth tests were commissioned (Stantec, 2006) to asses the toxicity of MEA and DEA to three plant species, alfalfa (*Medicago sativa*), barley (*Hordeum vulgare*), and northern wheatgrass (*Elymus lanceolatus*). Environment Canada (2005a) toxicity test protocols were used for this work with minor modifications as documented in Stantec (2006). The results are summarized in Tables A-4 (MEA) and B-4 (DEA). IC_{25} values for various endpoints for these three species ranged from 584 mg/kg to 2,250 mg/kg (MEA) and 858 mg/kg to 4,028 mg/kg (DEA). These data are analyzed in more detail in Section 11.1.

5.2 Soil Invertebrates

No data were found in the literature on the toxicity of MEA or DEA to terrestrial invertebrates. Accordingly, chronic survival and reproduction tests were commissioned (Stantec, 2006) for two invertebrate species, the earthworm (*Eisenia andrei*), and the springtail (*Folsomia canadida*). Environment Canada (2004, 2005b) toxicity test protocols were used for this work with minor modifications as documented in Stantec (2006). The results are summarized in Tables A-5 (MEA) and B-5 (DEA). IC_{25} values for reproduction endpoints for these two invertebrates ranged from 759 mg/kg to 2,016 mg/kg (MEA) and 171 mg/kg to 2,304 mg/kg (DEA). These data are analyzed in more detail in Section 11.1.

5.3 Soil Microbial Processes

No studies on the effects of the Amines on soil microbial processes were identified.

6. TOXICOLOGICAL EFFECTS IN MAMMALIAN SPECIES

Human health toxicological reference values (e.g., tolerable daily intake (TDI) or reference dose (RfD)) have not been established for MEA or DEA by Health Canada (2004), the U.S. EPA (2010a) or other regulatory agencies (Section 2.5). The AENV (2009a) protocol for developing soil and groundwater quality guidelines for these chemicals protective of human health requires a TDI. This section of the report has three primary objectives: i) to provide a general overview of the mammalian toxicology of the Amines[1]; ii) to provide a more detailed discussion of the repeated dose (sub-chronic, chronic, and reproductive) toxicity data relevant to oral exposure; and iii) to develop proposed TDIs for MEA and DEA with supporting rationale.

Toxicological data from all routes of exposure have been compiled in Tables A-6 and B-6 for MEA and DEA, respectively. However, the oral route of exposure is emphasized in this Section for the following reasons:

1. The inhalation pathway is not significant under environmental conditions due to low Henry's law constants, high water solubility, and significant binding to clays (Table 2) .
2. The oral route of exposure is important in the development of soil and groundwater quality guidelines protective of human health.

The toxicity of MEA and DEA to mammalian species via oral administration is illustrated in Figure 3. Mortality data are presented on the first line of each chart, and each symbol represents an LD_{50} value. Systemic data (chronic and sub-chronic oral) and reproduction data are presented on the second and third lines of each chart. For systemic and reproduction data, a hollow symbol indicates a test concentration at which no effects were seen, and a solid symbol indicates a test concentration at which effects were seen.

6.1 Metabolism, Distribution, and Elimination

Oral administration of ^{14}C-DEA resulted in nearly complete absorption from the gastrointestinal tract (NTP, 1992). Intravenous administration of ^{14}C-DEA to rats indicated that 28% of the dose was excreted in the urine within 48 hours, with very little being lost in either feces or expired breath. The remaining portion of the dose was retained in tissues, with the greatest concentrations residing in the liver and kidneys (NTP, 1992). The potential for DEA to bioaccumulate in tissues was investigated via an 8 week repeat exposure study. The results of this study suggested that DEA-derived radioactivity accumulated in tissues and reached steady state levels in approximately 4 weeks. Following exposure, DEA was eliminated with a half-life of approximately 1 week (NTP, 1992).

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[1] The interested reader is referred to Knaak et al. (1997) for a more detailed review of the mammalian toxicology of the Amines.

Available data for MEA indicated that most MEA accumulated in the liver, followed by the heart and brain.

6.2 Acute Toxicity

MEA

The single dose lethality of MEA has been studied in rats, mice, rabbits, and guinea pigs. LD_{50} values for oral exposure range from 600 to 15,000 mg/kg-bw/day (Table A-6).

DEA

The single dose lethality of DEA has been studied in rats, mice, rabbits, and guinea pigs. LD_{50} values for oral exposure are generally similar to MEA, and range from 700 to 3,300 mg/kg-bw/day (Table B-6). Apart from mortality, most of the toxicological observations noted in acute studies relate to effects on the liver and kidneys.

6.3 Dermal and Ocular Irritancy

MEA

MEA has been shown to be a moderate to severe eye, skin, and respiratory irritant in laboratory animals (Weeks et al., 1960; Haseman et al., 2005). However, in humans, MEA has been shown not to injure the skin in low concentrations (Klain et al., 1985), and is also a normal tissue metabolite as well as an essential component of tissue phospholipids (Dawson, 1957). Browning (1953) observed that when undiluted MEA is applied to human skin on gauze for 1 ½ hours, only marked redness and absorption of the skin result.

DEA

The undiluted liquid and 40% solutions produce severe eye burns, whereas a 15% solution produces only minor damage (Carpenter and Smyth, 1946). A 10% solution applied to rabbit skin caused redness; higher concentrations caused increasing injury (Carpenter and Smyth, 1946).

6.4 Sub-Chronic and Chronic Toxicity - Oral

MEA

Limited data were available on the chronic and sub-chronic oral toxicity of MEA.

Wernick et al. (1975) investigated the chronic toxicity of a mixture of hair dyes and chemicals used as mixing bases in dog food. The mixture included MEA at a proportion of 22.42%. In a chronic feeding test, beagle dogs were exposed to the composite at concentrations of 0, 19.5, and 97.5 mg/kg-bw/day (0, 4.4, and 22 mg/kg-bw/day as MEA) for 2 years. No significant dose-related effects were seen in any of the parameters studied, including survival, body weight, a range of blood and urine parameters, and organ weights. No gross or microscopic changes were seen in the various organs or tissues. No ultrastructural changes were seen in electron microscopic studies on sections of liver or urinary bladder. Overall, this study identified no significant dose-related findings in any of the parameters examined. Thus, the NOAEL from this study is 22 mg/kg-bw/day. However, it should be noted that because no toxicological effects were found at any of the doses used in the study in that there is no corresponding lowest observed adverse effect level (LOAEL) at which adverse effects were seen.

Smyth et al. (1951) exposed rats to MEA in their feed for 30 days as part of a series of range-finding studies. The doses ranged from 160 to 2,670 mg/kg-bw/day. Only limited, summary information is available from these studies. The authors reported "altered" liver or kidney weights in the 640 mg/kg-bw/day and higher dose groups, "microscopic lesions" (presumed to be in the liver and/or kidney), and death at doses of 1,280 mg/kg-bw/day and higher. Other endpoints that were probable but not found were reduced growth and reduced appetite. The NOAEL was 320 mg/kg-bw/day.

DEA

There were two early studies, and one more recent, definitive, study available on the oral sub-chronic toxicity of DEA.

Smyth et al. (1951) exposed rats to DEA in their feed for 30 days as part of a series of range-finding studies. Administered doses were 0, 5, 20, 90, 170, 350, and 680 mg/kg-bw/day. Only limited, summary information is available from these studies. The authors reported "altered" liver or kidney weights in the 90 mg/kg-bw/day dose groups, "microscopic lesions" (presumed to be in the liver and/or kidney), and death at doses of 170 mg/kg-bw/day and higher. Other endpoints that were probable but not found were reduced growth and reduced appetite. The NOAEL was 20 mg/kg-bw/day.

Hartung et al. (1970) administered 4,000 ppm DEA to rats in their drinking water as a neutralized solution for 7 weeks. Little experimental detail is available, but reported toxicological effects at this concentration included a pronounced normocytic anaemia without bone marrow depletion or increase in the number of reticulocytes, liver and kidney damage, and mortality.

NTP (1992) conducted a range of studies in which male and female F344/N rats and B6C3F$_1$ mice were exposed to DEA in their drinking water for 2 or 13 weeks. Parts of these studies were also published separately as Hejtmancik et al. (1987a,b) and Melnick et al. (1994a,b). The experimental design (species, number of animals per concentration, drinking water concentration, and test durations) are summarized below:

Species	*# Animals per Conc.*	*Drinking Water Concentrations (ppm)*	*Test Duration (weeks)*
F344/N rats (male)	5	0, 630, 1250, 2500, 5000, 10000	2
F344/N rats (female)	5	0, 630, 1250, 2500, 5000, 10000	2
B6C3F$_1$ mice (male)	5	0, 630, 1250, 2500, 5000, 10000	2
B6C3F$_1$ mice (female)	5	0, 630, 1250, 2500, 5000, 10000	2
F344/N rats (male)	10	0, 320, 630, 1250, 2500, 5000	13
F344/N rats (female)	10	0, 160, 320, 630, 1250, 2500	13
B6C3F$_1$ mice (male)	10	0, 630, 1250, 2500, 5000, 10000	13
B6C3F$_1$ mice (female)	10	0, 630, 1250, 2500, 5000, 10000	13

In the rat studies, the toxicological effect seen at the lowest concentration was typically microcytic anemia, indicated by dose-dependant decreases in erythrocyte and reticulocyte counts, mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH) and hemocrit. Other findings at higher concentrations included increased kidney weight, kidney damage (indicated by nephropathy, tubular epithelial necrosis, tubular mineralization, and changes in various urinalysis parameters), decreased testis and epididymis weight, demyelination of the brain and spinal chord, and death. The lowest LOAEL from any of the NTP (1992) rat studies was 160 ppm in the 13 week study on female rats for significantly decreased MCV and MCH and increased kidney weight. A toxicological effect was seen at the lowest dose used in the study, therefore there was no corresponding NOAEL. Decreases in MCV and MCH, while statistically significant, were changed only 2% and 0.5%, respectively, from controls. Kidney weight was increased 30% relative to controls. This LOAEL corresponded to a dose of approximately 14 mg/kg-bw/day.

In the mouse studies, the toxicological effect seen at the lowest concentration was typically increased liver weight and liver damage (indicate by hepatocellular cytologic alteration). Other findings at higher concentrations included hepatocellular necrosis, kidney weight increase and damage (indicated by nephropathy and tubular epithelial necrosis), relative heart weight increase and heart degeneration, cytologic alteration of the salivary gland, and death. The lowest LOAEL from any of the NTP (1992) mouse studies was 630 ppm in the 13 week study on male and female mice for significantly increased liver weight and liver damage (indicate by hepatocellular cytologic alteration). A toxicological effect was seen at the lowest dose used in the study, therefore there was no corresponding NOAEL. This LOAEL corresponded to a dose of approximately 104 mg/kg-bw/day in males, and 142 mg/kg-bw/day in females.

In a parallel series of dermal exposure experiments, NTP (1992) also exposed male and female F344/N rats and B6C3F$_1$ mice to DEA in their drinking water for 2 or 13 weeks. The experimental design (species, number of animals per concentration, target dose, and test durations) are summarized below:

Species	***Animals /Dose***	***Target Dose (mg/kg/d)***	***Test Duration (weeks)***
F344/N rats (male and female)	5	0, 125, 250, 500, 1000, 2000	2
B6C3F$_1$ mice (male and female)	5	0, 160, 320, 630, 1250, 2500	2
F344/N rats (male and female)	10	0, 32, 63, 125, 250, 500	13
B6C3F$_1$ mice (male and female)	10	0, 80, 160, 320, 630, 1250	13

Other than the development of skin lesions at the application site in the dermal tests, the application sites affected in each species were identical in the dermal studies and the corresponding drinking water studies.

6.5 Sub-Chronic and Chronic Toxicity - Inhalation

MEA

As noted at the beginning of Section 6, the inhalation pathway is of little direct relevance to the environmental toxicity of MEA. However, several studies on the inhalation toxicity of MEA have been completed. These studies primarily confirm the status of MEA as an dermal and respiratory irritant under the tested conditions. Most studies administer MEA as an aqueous aerosol, and achieve MEA concentrations in air greater than even the theoretical maximum concentration in air that could be obtained in equilibrium with pure phase MEA based on the vapour pressure. However, some of these studies also comment on systemic effects, and lend support to the liver and kidney being the primary target organs for the systemic toxicity of MEA. It is these systemic effects that are the main focus of this section.

Treon et al. (1957) exposed dogs, cats, guinea pigs, rats, and mice to concentrations of MEA vapour up to 793 mg/m^3. This concentration exceeds the vapour pressure of pure phase MEA at ambient conditions, and was achieved by means of an aerosol. Various signs of respiratory distress, but no systemic effects were noted.

Weeks et al. (1960) exposed dogs, rats, and guinea pigs to varying concentrations of highly purified MEA vapour using essentially continuous exposure conditions (23.5 hr/day, 7 days/week). The species, exposure concentration, and test durations are summarized below:

Species	***Exposure Concentration (ppm)***	***Test Duration (days)***
CFW rats (male and female)	5	40
CFW rats (male and female)	12	90
CFW rats (male and female)	66	24
Hartley guinea pigs (male)	15	24
Hartley guinea pigs (male)	75	24
Beagle dogs (male)	6	60
Beagle dogs (male)	12	90
Beagle dogs (male)	26	90

All high dose groups exhibited skin and respiratory irritation, and behavioural changes which were attributed to extreme sensitivity resulting from the irritancy. Systemic effects noted in high dose groups included a range of microscopic level effects on liver and kidney tissue.

Timofievskaya (1962) reported on a Soviet study in which rats were exposed to MEA (technical grade, 75% purity) at 80 to 160 ppm for 5 hr/day for 6 months. As reviewed by Binks et al. (1992), the authors identified liver and kidneys as target tissues for inhaled MEA in rats, but did not specify a known effect level.

Taken together, these inhalation studies show that aerosols of MEA can be significantly irritating to the skin and respiratory tract, and confirm the liver and kidney as primary target organs for systemic toxicity.

DEA

Two older studies were available on the inhalation toxicity of DEA. These studies are summarized in Table B-6, but are not discussed further here. The negligible vapour pressure and Henry's law constant for DEA (Table 2) make these studies of no relevance to developing environmental quality guidelines.

6.6 Reproduction and Developmental Toxicity

MEA

Wernick et al. (1975) investigated the reproductive toxicity of a composite of hair dyes and chemicals used as mixing bases in rat and rabbit feed. The composite included MEA at a proportion of 22.42%.

- In a reproduction and teratology test, Sprague-Dawley CD strain rats were exposed to the composite at concentrations of 0, 1,950 and 7,800 ppm in the diet (0, 34.5, and 138 mg/kg-bw/day as MEA) 8 weeks prior to mating, through gestation, and 21 days of lactation. In part I of the test, the females received the basal diet and the males received the test diet containing the composite dye material. In part 2, the males received the basal diet, while the females received the test diet. No significant dose-related effects were seen in any of the reproductive or teratological parameters observed, including male and female fertility, length of gestation, number of females with resorption sites, live pups per litter, pup body weights, or pup abnormalities.
- In a teratology test, female New Zealand White rabbits were exposed to the composite at concentrations of 0, 19.5, and 97.5 mg/kg-bw/day (0, 4.37, and 21.9 mg/kg-bw/day as MEA) on days 6-18 of gestation. Another group was exposed to the same doses of the composite without the dyes, resulting in slightly higher doses of MEA (0, 4.70, and 23.5 mg/kg-bw/day). No significant dose-related effects were seen in any of the teratological parameters observed, including fetal survival, gross fetal abnormalities, or soft tissue or skeletal fetal abnormalities.

Overall, this study identified no significant dose-related findings in any of the parameters examined. Thus, MEA NOAELs of 21.9 and 23.5 mg/kg-bw/day can be determined. However, it should be noted that because no toxicological effects were found at any of the doses used in the study, there is no corresponding LOAEL at which adverse effects were seen.

Mankes (1986) investigated the toxicity of MEA on the development of rat embryos. In this teratological study, pregnant Long-Evans rats were dosed with MEA by gavage on days 6 to 15 of gestation, the so-called "critical period" of organogenesis. Study results were evaluated at day 20 of gestation, at which point the dams were euthanized, and the pups delivered by caesarean section. The administered doses were 0, 50, 300, and 500 mg/kg-bw/day (0, 2.4%, 14.4% or 24% of the LD_{50} value). There were 8-10 rats in each dose group, and 34 in the control group. At the 500 mg/kg-bw/day dose, increased maternal toxicity and embryolethality were observed. At the 300 mg/kg-bw/day dose, some pups showed significant reductions in body weight and increases in malformation rate. At the lowest dose rate, 50 mg/kg-bw/day, malformation rates (hydronephrosis and sternebral variations) were increased only in male offspring that were contiguous in the uterus with one or more male siblings. Hydronephrosis is an obstruction of the free flow of urine from the kidney. Sternebral variations are differences in the four segments of the sternum. In summary, this study identified 500 mg/kg-bw/day as the embryolethal and maternal toxicity dose, 300 mg/kg-bw/day as the embryotoxic dose, and 50 mg/kg-bw/day as the LOAEL for developmental effects for Long-Evans rats exposed to MEA during day 6 to 15 of gestation. A toxicological effect was seen at the lowest dose used in the study, therefore there there is no corresponding NOAEL.

Pereira et al. (1987). This unpublished report was referenced and summarized in Hellwig and Liberacki (1997), but a copy was not obtained for review in the current project. Information presented below is repeated from Hellwig and Liberacki (1997). Pereira et al. (1987) tested MEA using the Chernoff–Kavlock postnatal mouse screening assay. In brief, this assay measures embryonic, fetal, and neonatal toxic responses following high dose exposure (1 dose level) of pregnant mice treated during the period of major organogenesis and is primarily used to set priorities for further testing. In this assay, oral administration of 850 mg MEA/kg-bw/day to pregnant CD-1 mice on days 6– 15 of gestation resulted in 16% mortality of maternal animals and reduced numbers of viable litters. Administration of MEA did not affect litter size, percentage survival of pups, birth weight, or weight gain of pups.

Liberacki (1996) investigated the toxicity of MEA on the development of rat and rabbit embryos via dermal exposure. Pregnant Sprague-Dawley rats and pregnant New Zealand white rabbits were exposed dermally to MEA at 0, 10, 25, and 75 mg/kg-bw/day. A high dose group of rats (but not rabbits) were exposed dermally to 225 mg/kg-bw/day. Exposure was conducted for approximately 6 hours per day on days 6 through 15 (rats) and 6 through 18 (rabbits) of gestation. Dermal exposure of pregnant rats to 225 mg/kg-bw/day and rabbits to 75 mg/kg-bw/day resulted in significant increases in the incidence of skin irritation/lesions and maternal body weight effects. Doses of 25 mg/kg/day to rabbits produced only minor irritation. Despite maternal effects observed in rats and rabbits, no evidence of developmental or fetal toxicity was observed at any dose level tested. Thus, it was concluded that MEA was not developmentally toxic following dermal application at exposure levels up to and including 225 mg/kg/day for rats and 75 mg/kg for rabbits.

Hellwig and Liberacki (1997) also investigated the toxicity of MEA on the development of rat embryos. Their study was conducted to meet the requirements of Good Laboratory Practice (GLP) for the Organization of Economic Co-operation and Development (OECD). In this teratological study, pregnant Wistar rats were dosed with MEA by gavage on days 6 to 15 of gestation, the so-called "critical period" of organogenesis (40 rats per group). Study results were evaluated i) at day 20 of gestation (25 dams per group), at which point the dams were euthanized, and the pups delivered by caesarean section, and ii) at day 21 postpartum, at which point dams and pups were euthanized and examined. The administered doses were 0, 40, 120, and 450 mg/kg-bw/day. Evidence of maternal toxicity was seen in the 450 mg/kg-bw/day group, but not the 40 or 120 mg/kg-bw/day groups. Despite the maternal toxicity seen at 450 mg/kg-bw/day, no significant fetal effects were observed at this or any dose level tested, nor were there any indications of a treatment-related effect on postnatal growth or the viability of offspring. The findings of this study are in apparent contrast to Mankes (1986) who found fetal effects at doses as low as 50 mg/kg-bw/day. Hellwig and Liberacki (1997) note this discrepancy, but point out that in the Mankes (1986) report "an atypical classification scheme was used, which classified runting, hydroureter and unspecified skeletal alterations as malformations rather than developmental variations, as is more common practice". The study concluded that MEA was not developmentally toxic to Wistar rats following repeated oral administration, even at maternally toxic levels.

DEA

NTP (1999a) investigated the developmental toxicity of DEA on rats. In this study, pregnant Sprague-Dawley rats were dosed with DEA by gavage on days 6 through 19 of gestation. Maternal condition was evaluated on post natal day 21. Naturally delivered offspring were monitored for clinical condition on post natal days 0, 4, 7, 14, and 21. The administered doses were 0, 50, 125, 200, 250 and 300 mg/kg-bw/day. Maternal effects included: increased kidney weight and altered water intake at or above 125 mg/kg/d; reduced body weight gain and altered feed intake at or above 200 mg/kg/d. Effects on offspring included: increased early post natal mortality at or above 125 mg/kg/d; post-implantation mortality was increased and pup body weight was decreased at or above 200 mg/kg/d. Overall, therefore, the LOAEL was 125 mg/kg/d and the NOAEL was 50 mg/kg/d for both maternal and developmental toxicity.

6.7 Carcinogenicity and Genetic Toxicity

MEA

No studies relevant to the assessment of the carcinogenicity of MEA were found. The National Toxicology Program (NTP) has not conducted studies on the carcinogenicity of MEA.

MEA has been demonstrated to be nonmutagenic in the Ames *Salmonella typhimurium* assay, with and without S9 microsomal metabolic activation, using strains TA1535, TA1537, TA1538, TA98, and TA100; and also negative in the *E. coli* assay, *Saccharomyces* gene conversion assay, and rat liver chromosome assay (Dean et al., 1985).

In summary, there is no evidence that MEA causes carcinogenicity or genetic toxicity.

DEA

NTP (1999b) conducted 2 year carcinogenicity studies on the dermal application of DEA in an ethanol carrier to F334/N rats and B6C3F$_1$ mice. Groups of 50 male rats were administered 0, 16, 32, or 64 mg of DEA/kg body weight in ethanol dermally for 2 years. Groups of 50 female rats were administered 0, 8, 16, or 32 mg of DEA/kg body weight in ethanol dermally for 2 years. Groups of 50 male and 50 female mice were administered 0, 40, 80, or 160 mg of DEA/kg body weight in ethanol dermally for 2 years.

NTP (1999b) found no evidence of carcinogenic activity of DEA in male or female F344/N rats. However, reported a range of carcinogenic effects on the liver, kidney and other organs in mice. Endpoints noted included increased incidence of liver neoplasms in males and females and increased incidence of renal tubule neoplasms in males. The overall conclusion of the NTP (1999b) report was that that there was clear evidence of carcinogenic activity of DEA in male and female $B6C3F_1$ mice under the conditions tested. However, various reviewers from the report's Technical Review Subcommittee had concerns with certain aspects of the findings (NTP, 1999b). Dr. John Bailer commented on the high liver neoplasm rates in control mice in this study, and pointed out that the historical control database is small for dermal studies using an ethanol vehicle. Dr. Linda Chapman did not agree with the conclusions for mice, stating that DEA is not a mutagen and is not metabolized to a reactive intermediate, but can be converted to a carcinogenic nitrosamine. She felt that the potential for N-nitroso-diethanolamine formation should have been evaluated. Dr. Stephen Hecht stated his disappointment with the lack of detail in the analytical methods description so that contamination of the DEA with N-nitroso-diethanolamine could not be ruled out. In addition, it has been proposed (Jon Busch, Director, American Chemistry Council, pers. comm., 2001) that the NTP (1999b) study was flawed, in that neoplasms could have been a result of the ethanol carrier used for DEA. Ethanol can cause choline deficiency which in turn can cause tumors in rodents.

DEA was not mutagenic in any of four strains of *Salmonella typhimurium*, in the presence or absence of S9 metabolic activation enzymes. No induction of trifluorothymidine resistance was observed in L5178Y mouse lymphoma cells treated with DEA with or without S9. DEA did not induce significant sister chromatid exchanges or chromosomal aberrations in cultured Chinese hamster ovary cells, with or without S9. Peripheral blood samples collected from male and female mice exposed to 80 to 1,250 mg/kg DEA dermally for 13 weeks showed no increase in micronucleated normochromatic erythrocytes (NTP, 1999b).

In summary, there is no evidence that DEA causes carcinogenicity in rats, there is evidence of carcinogenicity in mice, which may be confounded, and there is no evidence that DEA causes genetic toxicity.

6.8 Odour Threshold

Weeks et al (1960) reported that the odour threshold at which human subjects could detect an odour from MEA was 2.6 ppm (12 subjects).

No odour threshold data were found for DEA.

6.9 Summary of Toxicity and Proposed Tolerable Daily Intake

MEA

In the absence of any indications of carcinogenicity or mutagenicity, MEA is treated as a threshold toxicant.

The toxicology of MEA was discussed in the preceding sections, and key points may be summarized as follows:

- MEA is a moderate to severe eye, skin, and respiratory irritant.
- The oral LD_{50} of MEA ranges from 600 to 15,000 mg/kg-bw/day in various test species.
- The lowest LOAEL for chronic or sub-chronic systemic effects was 640 mg/kg-bw/day from an early study (Smyth, 1951), which found effects on kidney and liver organ weights in a 30 day rat study at this dose level. The corresponding NOAEL was 320 mg/kg-bw/day.
- A chronic (2 year) study on dogs found no effects at 22 mg/kg-bw/day (no toxic effects seen at any dose used in the study).
- The dataset on reproductive and teratological effects (5 studies: 4 oral, one dermal) is inconsistent. All studies with sufficiently high doses observed maternal toxicity at 450 to 850 mg/kg-bw/day. However, one study found reproductive/teratological effects at all doses tested (50, 300, and 500 mg/kg-bw/day), while the other 4 studies found no reproductive/teratological effects at any dose tested, including, in some cases, doses high enough to cause maternal toxicity.

Before a final TDI could be developed for MEA, i) a definitive modern study on the chronic or sub-chronic systemic effects of MEA via oral exposure; and ii) resolution of the discrepancy between the Mankes (1986) and the other reproduction/teratological studies would be required.

However, for the present, an interim TDI is proposed that uses the precautionary principle with the existing dataset. The precautionary principle would indicate that the results of the Mankes (1986) study should be taken at face value, in spite of the conflicting evidence of four other studies. If this is done, then the lowest LOAEL from any study is 50 mg/kg-bw/day from Mankes (1986). Normally, the NOAEL associated with the lowest relevant LOAEL would be used as the departure point for calculating a TDI. A toxicological effect was seen at the lowest dose used in the study, therefore there is no NOAEL. However, Health Canada acknowledges that it may sometimes be necessary to calculate a TDI based on a LOAEL that has no associated NOAEL with the use of appropriate additional safety factors (Wilson and Orr, 2004). In this case, it is noted that 3 other studies found no reproductive effects at doses significantly greater than the LOAEL of 50 mg/kg-bw/day, and this LOAEL is used as the departure point for developing a TDI.

Uncertainty Factors and Calculation of TDI

The following uncertainty factors are proposed (consistent with Wilson and Orr, 2004):

- A factor of 10 to account for interspecies differences.
- A factor of 10 to account for intraspecies (inter-individual) differences.
- A factor of 10 to account for the aggregate of limitations and inconsistencies in the dataset and the fact that the point of departure is a LOAEL, rather than a NOAEL.

Thus the overall uncertainty factor is 1,000, and the TDI is calculated by dividing the point of departure by the uncertainty factor to give a TDI of 0.05 mg/kg-bw/day. This is the TDI used in calculating soil and groundwater guidelines for human exposure pathways in this document (Table 3).

DEA

Carcinogenicity

- There is no indication of carcinogenicity in rats, and no indication of mutagenicity in any species tested. There were indications of carcinogenicity in mice, however, the significance of these findings has been disputed by a number of reviewers. It has been suggested that the findings in mice may have been confounded in that the neoplasms observed could have been a result of the ethanol carrier used for DEA.
- Health Canada (2004) has not classified DEA for carcinogenicity. However, considering the criteria for classification provided in Health Canada (1994), it appears that the dataset (no evidence of carcinogenicity in rats, equivocal/disputed evidence in mice, and no evidence of mutagenicity/genotoxicity) is consistent with classification in Group III ("Possibly Carcinogenic to Humans"). The definition for Group III D includes chemicals for which the "data from experimental studies in animal species indicate that the compound is carcinogenic in one species only and there is suspicion that the results are species-specific but available data on mechanisms of toxicity are insufficient to conclude unequivocally that this is the case". Statements in the definitions of Groups III B and III C also appear to have relevance for the available DEA dataset.
- It is further noted that the lowest LOAEL for non-carcinogenic effects in DEA (14 mg/kg/d) was lower than the lowest dose in the mouse carcinogenicity study.
- Considering the weight of available evidence, for the purposes of the current document, DEA was treated as a Health Canada (1994) Group III carcinogen.
- Wilson and Orr (2004) indicate that, for Group III carcinogens, a cancer potency is generally not derived. Instead, an additional uncertainty factor to account for uncertainty

in the potential for human carcinogenicity, is applied to establish an interim TDI or threshold concentration (TC).

Summary of Non-Carcinogenic Toxicity

- DEA is a mild eye and skin irritant at low concentrations (~5%), and a more significant irritant at higher concentrations.
- The oral LD_{50} of DEA ranges from 700 to 3,300 mg/kg-bw/day in various test species.
- The lowest LOEL for systemic effects was 160 ppm in a 13 week drinking water study on female rats. Effects identified at this concentration were significantly decreased MCV and MCH and increased kidney weight. Decreases in MCV and MCH, while statistically significant, were changed only 2% and 0.5%, respectively, from controls. Kidney weight was increased 30% relative to controls. This LOAEL corresponded to a dose of approximately 14 mg/kg-bw/day. A toxicological effect was seen at the lowest dose used in the study, therefore there was no corresponding NOAEL.
- A study on the reproductive toxicity of DEA to rats found a LOAEL of 125 mg/kg/d for both maternal toxicity (increased kidney weight and altered water intake) and developmental toxicity (increased early post natal mortality). The corresponding NOAEL was 50 mg/kg/d.

The lowest LOAEL from any study is 14 mg/kg/d from NTP (1992). Normally, the NOAEL associated with the lowest relevant LOAEL would be used as the departure point for calculating a TDI. A toxicological effect was seen at the lowest dose used in the study, therefore there is no associated NOAEL. However, Health Canada acknowledges that it may sometimes be necessary to calculate a TDI based on an LOAEL that has no associated NOAEL with the use of appropriate additional safety factors (Wilson and Orr, 2004). This LOAEL of 14 mg/kg-bw/day is used as the departure point for developing a TDI.

Uncertainty Factors and Calculation of TDI

The following uncertainty factors are proposed (consistent with Wilson and Orr, 2004):

- A factor of 10 to account for interspecies differences.
- A factor of 10 to account for intraspecies (inter-individual) differences.
- A factor of 10 to account for the point of departure being a LOAEL which has no associated NOAEL from a sub-chronic study.
- An additional factor of 3 to account for uncertainty in the carcinogenicity database.

Thus, the overall uncertainty factor is 3,000, and the TDI is calculated by dividing the point of departure (14 mg/kg/d) by the uncertainty factor of 3,000 to give a TDI of 0.005 mg/kg-bw/day. This is the TDI used in calculating soil and groundwater guidelines for human exposure pathways in this document (Table 3).

7. DATA ADEQUACY AND DATA GAPS

The available data were assessed against AENV (2009a) and CCME (2006) requirements for developing soil and water guidelines.

7.1 Human Health Guidelines

In the absence of regulatory toxicity reference values, human health guidelines for the direct contact and protection of potable groundwater pathways and source guidance values for groundwater were calculated based on the tolerable daily intake values developed in this document. The toxicological datasets for MEA and DEA are extensive, but include significant complexities and potential contradictions. The TDIs developed in this document took a conservative approach to reflect these dataset complexities. There is scope for further, definitive, toxicological studies that would resolve some of these issues, and could potentially result in changing one or both TDI values.

Guidelines protective of indoor air inhalation are not required and were not calculated, since the Amines have very low vapour pressures and Henry's law constants.

Guidelines protective of ingestion of produce, milk and meat are not required and were not calculated, since the Amines are not expected to biomagnify, based on their BCF values.

7.2 Ecological Guidelines

Additional data (Stantec, 2006) were commissioned to fulfil the dataset required to develop soil remediation guidelines for the eco-contact pathway.

None of the available data are suitable for calculating the nutrient and energy cycling check. Consistent with the CCME (2006) protocol, a soil remediation guideline was calculated without this check. However, if it was desired to calculate this check, it would be necessary to conduct a minimum of three microbial process studies, ideally considering nitrification and nitrogen-fixation endpoints.

Additional data (Vizon, 2006) were commissioned to fill data gaps in the (CCME, 1991) minimum required dataset to calculate interim freshwater aquatic life water quality guidelines. Further tests, including chronic fish and invertebrate tests, would be required to fulfil all the requirements for full freshwater aquatic life water quality guidelines.

Insufficient data exist to calculate soil and food ingestion guidelines. The CCME (2006) protocol for this guideline requires toxicity data from tests conducted on livestock species, and these data do not currently exist for MEA and DEA. This data gap is not considered particularly significant, since the MEA and DEA are not expected to bioconcentrate significantly into fodder.

Insufficient data exist to calculate irrigation water guidelines. The minimum data requirement (CCME, 1993) for developing an interim irrigation guideline is two studies on cereal, tame hay, or pasture crops, and two studies on other crops. An irrigation water guideline was not calculated. However, this data gap is not considered particularly significant, since the MEA and DEA are expected to degrade rapidly in surface soil and are not expected to bioconcentrate into plants.

Insufficient data are available to meet the requirements published in CCME (1993) for developing a livestock watering guideline; therefore, this guideline was not calculated.

8. PARAMETER VALUES

Parameter values required to calculate Alberta Tier 1 soil and groundwater remediation guidelines for MEA and DEA fall into two main groups: i) parameters that relate to the chemical properties, toxicity, or background exposure to the Amines, referred to as "chemical-specific parameters"; and, ii) parameters relating to receptor exposure and properties of the site, referred to as "non-chemical-specific parameters". These two groups of parameters are discussed below.

8.1 Chemical-Specific Parameters

Chemical-specific parameters for MEA and DEA are summarized in Table 3, together with an indication of where to find a discussion of the rationale for the value selected. The soil allocation factor (SAF) and water allocation factor (WF) each take the values of 0.25 (Table 3), since exposure to MEA and DEA could reasonably be anticipated via four potentially contaminated environmental media: soil, water, food, and consumer products. However, exposure via air, the fifth potentially-contaminated medium, is unlikely due to the negligible vapour pressure of the Amines (Section 2.5).

8.2 Non Chemical-Specific Parameters

Non chemical-specific parameter values are taken without change from AENV (2009a). Parameter values for human receptor characteristics, soil and hydrogeological parameters, site characteristics, and building parameters are provided in Tables 4 to 7, respectively.

9. SURFACE WATER GUIDELINES

AENV (2009a) and CCME (2006) use surface water quality guidelines as a basis from which to calculate corresponding groundwater and soil remediation guidelines. Surface water quality guidelines calculated for MEA and DEA are provided and discussed below.

9.1 Human Drinking Water

No Canadian Drinking Water Quality Guideline (CDWQG) currently exists for any of the Amines. In such cases, CCME (2006) includes a protocol for calculating an allowable concentration in potable water (Source Guidance Value for Groundwater) from the tolerable daily intake using the following equation:

$$SGVG = \frac{TDI \times BW \times WF}{WIR}$$

where:

SGVG = Source Guidance Value for Groundwater (mg/L)
TDI = tolerable daily intake (mg/kg/d)
BW = body weight (kg)
WF = water allocation factor (unitless)
WIR = water ingestion rate (L/d)

The SGVG is calculated using adult parameters (CCME, 2006). Substituting appropriate parameter values from Tables 3 and 4 gives values of 0.59 mg/L (MEA) and 0.059 mg/L (DEA). These values are rounded to 1 significant figure with a 5 or 0 in the second figure to give 0.6 mg/L (MEA) and 0.06 mg/L (DEA) which are the Source Guidance Values for Groundwater for these compounds (Table 8).

9.2 Freshwater Aquatic Life

Interim freshwater aquatic life water quality guidelines for MEA and DEA were calculated based on the CCME (1991) protocol. Freshwater aquatic toxicity data were obtained from the U.S. EPA (2010b) ECOTOX database and other sources discussed in Section 4, and are summarized in Tables A-2 and B-2, for MEA and DEA respectively.

Data Quantity Requirements

Insufficient data exist for the development of full freshwater aquatic life water quality guidelines for MEA or DEA. However, minimum data requirements are met for both chemicals for the development of an interim guideline (two acute and/or chronic studies on two or more fish species, including one cold water species resident in North America; two acute and/or chronic studies on two or more invertebrate species from different classes, including one planktonic species). Thus it was possible to develop interim freshwater aquatic life water quality guidelines for both MEA and DEA.

Data Quality Screening

Aquatic toxicological data were screened for data quality and assigned to Primary, Secondary, or Unacceptable categories, based on the CCME (1991) criteria. Initial data screening was completed based on information available in the U.S. EPA (2010b) ECOTOX database. Data were placed into the Unacceptable category for one of the following reasons:

- The effect was not ecologically relevant.
- No controls were included in the test design, or no information was provided on controls.
- No data were available on test duration.
- No data were available on the effect that was tested.
- The data point does not represent an effect (e.g. no observed effect concentration (NOEC) endpoint, or concentration given as greater than a certain value).
- Test media (e.g., fresh water, salt water, other) were not clearly identified.

Comments are provided in Tables A-2 and B-2 indicating the rationale for considering each study Unacceptable.

Guideline Calculation

Surface water guidelines for MEA and DEA were calculated using the CCME (1991) protocol which considers Primary and Secondary data and takes the lower of:

1. the lowest LOEC for a chronic study for a non-lethal endpoint is multiplied by a safety factor of 0.1.
2. The lowest EC_{50} or LC_{50} for an acute test is multiplied by an application factor of 0.05 (MEA and DEA are considered non-persistent in surface water as discussed in Section 3.4.3).

Details of the calculations for each chemical are provided below.

9.2.1 MEA

Primary and Secondary toxicity studies for MEA were reviewed in Section 4.1.1.

Chronic Studies

The lowest endpoint from a chronic study among the Primary and Secondary data in Table A-2 is 0.75 mg/L which is the Bringmann and Kuhn (1980) LC_{03} for growth inhibition in the green alga (*Scenedesmus quadricauda*). A freshwater aquatic life water quality guideline based on this chronic study was calculated by multiplying the LC_{03} of 0.75 mg/L from this study by a safety factor of 0.1 to give a guideline value of 0.075 mg/L.

Acute Studies

The freshwater guideline derived from the lowest relevant acute EC_{50}/LC_{50} is calculated by multiplying the Vizon (2006) 48 hour LC_{50} for *Daphnia magna* (67 mg/L) by an application factor of 0.05 (non-persistent variable, Section 3.4.3) to give a guideline value of 3.35 mg/L.

The guideline value from the chronic study is the lower of the two values calculated above, and accordingly, the freshwater aquatic life water quality guideline for MEA is 0.075 mg/L (Table 8).

9.2.2 DEA

Primary and Secondary toxicity studies for MEA were reviewed in Section 4.1.2.

Chronic Studies

The lowest endpoint from a chronic study among the Primary and Secondary data in Table A-2 is 4.4 mg/L which is the Bringmann and Kuhn (1980) LC_{03} for growth inhibition in the green alga (*Scenedesmus quadricauda).* A freshwater aquatic life water quality guideline based on this chronic study was calculated by multiplying the LC_{03} of 4.4 mg/L from this study by a safety factor of 0.1 to give a guideline value of 0.44 mg/L.

Acute Studies

The freshwater guideline derived from the lowest relevant acute EC_{50}/LC_{50} is calculated by multiplying the Cowgill et al. (1985) 48 hour LC_{50} for *Ceriodaphnia dubia* (29 mg/L) by an application factor of 0.05 (non-persistent variable, Section 3.4.3) to give a guideline value of 1.45 mg/L.

The guideline value from the chronic study is the lower of the two values calculated above, and accordingly, the freshwater aquatic life water quality guideline for DEA is 0.44 mg/L. This value is rounded to 1 significant figure with a 5 or 0 in the second figure to give 0.45 mg/L (Table 8).

9.3 Irrigation Water

No guideline was calculated for the Amines in irrigation water, since the minimum data requirements were not met (Section 7.2).

9.4 Livestock and Wildlife Watering

Toxicity data for the Amines were not available for livestock or wildlife species (Section 7.2), and accordingly, these guidelines could not be calculated.

10. SOIL AND GROUNDWATER GUIDELINE CALCULATIONS – HUMAN HEALTH

10.1 Direct Contact

The model used to calculate the soil remediation guideline protective of the human direct soil contact (soil ingestion, dermal contact, and particulate inhalation) exposure pathway for the Amines is taken without change from AENV (2009a). Parameter values are summarized in Tables 3 and 4. The following equation was used.

$$PSQG_{HH} = \frac{(TDI - EDI) \times SAF \times BW}{[(AF_G \times SIR) + (AF_L \times IR_S \times ET_2) + (AF_S \times SR)] \times ET_1} + [BSC]$$

Where:

$PSQG_{HH}$ = preliminary human health-based soil remediation guideline (mg/kg)
TDI = tolerable daily intake (mg/kg-bw/day)
EDI = estimated daily intake (mg/kg-bw/day)
SAF = soil allocation factor (dimensionless)
BW = adult or toddler body weight (kg)
AF_G = absorption factor for gut (dimensionless)
AF_L = absorption factor for lung (dimensionless)
AF_S = absorption factor for skin (dimensionless)
SIR = adult or toddler soil ingestion rate (kg/day)
IR_S = inhalation of particulate matter re-suspended from soil (kg/day)
SR = adult or toddler soil dermal contact rate, see below (kg/day)
ET_1 = exposure term 1 (dimensionless) (days/week ÷ 7 x weeks/year ÷ 52)
ET_2 = exposure term 2 (dimensionless) (hours/day ÷ 24)
BSC = background soil concentration (mg/kg)

Substituting appropriate values from Tables 3 and 4 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives human direct contact guideline values of:

MEA (Tables 9 and 10):

- 1,500 mg/kg (agricultural and residential);
- 2,000 mg/kg (commercial); and,
- 10,000 mg/kg (industrial).

DEA (Tables 11 and 12):

- 150 mg/kg (agricultural and residential);
- 200 mg/kg (commercial); and,

- 1,000 mg/kg (industrial).

Soil Dermal Contact Rate

The soil dermal contact rate (SR) is the mass of contaminated soil which is assumed to contact the skin each day. This parameter is calculated as follows (AENV, 2007a):

$$SR = \{(SA_H \times DL_H) + (SA_O \times DL_O)\} \times EF$$

Where:

SR = soil dermal contact rate (kg/day)
SA_H = exposed surface area of hands (m^2)
DL_H = dermal loading of soil to hands (kg/m^2 per event)
SA_O = area of exposed body surfaces other than hands (m^2)
DL_O = dermal loading of soil to other surfaces (kg/m^2 per event)
EF = exposure frequency (events/day)

The soil dermal contact rate is calculated separately for toddlers and adults using the parameters in Table 4, and is 6.88×10^{-5} kg/day for toddlers, and 1.14×10^{-4} kg/day for adults.

10.2 Inhalation

The Amines are effectively non-volatile (Table 2) and accordingly remediation guidelines protective of the indoor air inhalation exposure pathway are not required or calculated for either soil or groundwater.

10.3 Offsite Migration

Offsite Migration guidelines are calculated to check that the guidelines set for commercial and industrial land use will not result in adjacent, more sensitive land being contaminated at levels above the applicable guideline due to wind and/or water transport of contaminated soil from the commercial or industrial site. The human health offsite migration guideline is calculated using the equation provided in AENV (2009a):

$$SQG_{OM} = (14.3 \times SQG_A) - (13.3 \times BSC)$$

Where

SQG_{OM} = soil remediation guideline protective of offsite migration (mg/kg)
SQG_A = soil remediation guideline for human direct soil contact for agricultural land use (mg/kg)
BSC = background soil concentration (mg/kg)

Substituting appropriate values from Tables 3, 9, 10, 11, and 12 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives a human health offsite migration guideline of 20,000 mg/kg for MEA (Tables 9 and 10) and 2,000 mg/kg for DEA (Tables 11 and 12).

11. SOIL AND GROUNDWATER GUIDELINE CALCULATIONS – ECOLOGICAL

11.1 Direct Contact

11.1.1 Soil

The soil remediation guideline for the exposure pathway considering direct contact of plants and soil invertebrates (the "eco-contact pathway") was calculated for MEA and DEA based on a weight of evidence approach following CCME (2006). Data relevant for guideline development are sourced from Stantec (2006) and are summarized in Tables A-4 and A-5 (MEA) and B-4 and B-5 (DEA). The values provided in the above-noted tables are nominal values based on the known amount of chemical spiked into the test soils.

Stantec (2006) included analytical data to confirm exposure concentrations. Analytical recovery of the Amines from soil proved to be highly variable. A detailed study confirmed that analytical methods for the Amines were inadequate to quantify these compounds in soil with confidence. Subsequent to this work, an improved analytical method (see Appendix C) has been developed for the Amines. Due to the variability in the analytical results obtained concurrently with this toxicological study, the analysis below is based on nominal concentrations.

The CCME (2006) protocol uses data standardized at the 25th percentile effect level. Invertebrate survival data were not calculated at the 25% effect level by Stantec (2006), and were not included in the calculation of guideline values. Where wet mass and dry mass are provided separately in Stantec (2006), these endpoints are considered redundant, and only the dry mass data (generally considered to be more reliable) are included here. The data that were used to calculate the eco-contact guideline are presented below. These data have not been corrected for analytical recovery.

The 25th percentile of these data is the eco-contact guideline for natural areas, agricultural and residential. The 50th percentile of these data is the eco-contact guideline for commercial and industrial land use. The eco-contact guidelines for MEA and DEA are summarized below (rounded to 1 significant figure with a 5 or a 0 as the second figure) and included in Tables 9, 10, 11, and 12.

Species	Effect	IC$_{25}$ (Not Corrected for Analytical Recovery) MEA (mg/kg)	IC$_{25}$ (Not Corrected for Analytical Recovery) DEA (mg/kg)
Alfalfa	Shoot Length	1,460	1,194
Alfalfa	Root Length	1,611	2,109
Alfalfa	Shoot Dry Mass	862	995
Alfalfa	Root Dry Mass	584	1,077
Barley	Shoot Length	2,250	3,194
Barley	Root Length	1,473	4,028
Barley	Shoot Dry Mass	2,022	2,247
Barley	Root Dry Mass	1,557	858
Northern Wheatgrass	Shoot Length	1,626	3,290
Northern Wheatgrass	Root Length	2,107	3,575
Northern Wheatgrass	Shoot Dry Mass	1,201	1,602
Northern Wheatgrass	Root Dry Mass	1,918	2,204
Eisenia andrei	Number of Progeny	2,016	171
Eisenia andrei	Dry Mass of Individual Progeny	1,905	2,136
Folsomia candida	Number of Progeny	1,250	2,102

MEA

- 25th percentile - natural areas, agricultural and residential: 1,500 mg/kg.
- 50th percentile - commercial and industrial: 1,500 mg/kg.

DEA

- 25th percentile - natural areas, agricultural and residential: 1,000 mg/kg.
- 50th percentile - commercial and industrial: 2,000 mg/kg.

These guidelines apply to both coarse- and fine-grained soils.

11.1.2 Groundwater

The direct contact of shallow groundwater with plants and soil invertebrates exposure pathway is applicable whenever groundwater is present within 3 m of the ground surface. However, based on guidance in AENV (2009a), the guideline is not calculated for polar compounds such as the Amines. The rationale for this position is that the potential interactions between polar organic compounds and soils are complex in that they can be highly dependant on various environmental conditions including pH, clay mineralogy, and redox conditions. Attempting to set groundwater guidelines for polar chemicals for this pathway would involve significant uncertainty, and accordingly, it is recommended that concerns with potential adverse effects on surface soil biota from polar organic compounds in shallow groundwater be addressed on a site-specific basis by analyzing soil samples.

Accordingly, the groundwater guideline protective of the eco-contact pathway is not calculated for the Amines.

11.2 Nutrient and Energy Cycling

Insufficient data were available and this guideline was not calculated for the Amines.

11.3 Soil and Food Ingestion

Insufficient data were available (Section 7.2), and this guideline was not calculated for the Amines. However, this exposure pathway was not expected to be a concern, since i) the Amines are expected to degrade rapidly in surficial soil (Section 3.5) and accordingly livestock and wildlife are unlikely to get significant exposure to the Amines through incidental ingestion of surficial soil; and ii) based on their very low K_{ow} values (negative log K_{ow}; Table 2) MEA and DEA are not expected to accumulate into plants to any significant extent; thus, the exposure of livestock or wildlife to MEA and DEA in soil via ingestion of fodder is expected to be minimal.

11.4 Offsite Migration

Offsite Migration guidelines are calculated to check that the guidelines set for commercial and industrial land use will not result in adjacent more sensitive land being contaminated at levels above the applicable guideline for the sensitive land due to wind and/or water transport of contaminated soil from the commercial or industrial site. The ecological offsite migration guideline is calculated using the equation provided in AENV (2009a):

$$SQG_{OM} = (14.3 \times SQG_A) - (13.3 \times BSC)$$

Where SQG_{OM}= soil remediation guideline protective of offsite migration (mg/kg)
SQG_A = soil remediation guideline for ecological direct soil contact for agricultural land use (mg/kg)
BSC = background soil concentration (mg/kg)

Substituting appropriate values from Tables 3, 9, 10, 11, and 12 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives ecological offsite migration guidelines of 20,000 mg/kg for MEA (Tables 9 and 10), and 15,000 mg/kg for DEA (Tables 11 and 12).

12. SOIL AND GROUNDWATER GUIDELINE CALCULATIONS – GROUNDWATER PATHWAYS

This section provides the protocols used to calculate soil and groundwater remediation objectives protective of exposure pathways involving groundwater. The following receptors are considered:

- humans (potable drinking water sourced from groundwater); and,
- aquatic life (via lateral groundwater transport and discharge into a surface water body).

In the first case, it is assumed that a water well could potentially be installed at any location, and hence, it is assumed that there is no lateral offset between the location where the contaminated soil or groundwater is measured and the receptor.

In the second case, a minimum lateral separation of 10 m is assumed between the location where the contaminated soil or groundwater is measured and the location of the surface water body. In cases where contamination is present within 10 m of a surface water body, a site-specific approach will be required (see AENV, 2009b).

Surface water quality guidelines protective of the above water uses are provided in Table 8. As noted in Section 9, insufficient data are available to calculate surface water guidelines for the Amines protective of irrigation, wildlife or livestock watering, and accordingly, neither soil nor groundwater guidelines protective of these water uses could be calculated.

12.1 Soil Remediation Guidelines

Soil remediation guidelines for groundwater pathways were calculated using the model and equations from AENV (2009a)

12.1.1 Model Assumptions

Assumptions implicit in the model include the following:

- the soil is physically and chemically homogeneous;
- moisture content is uniform throughout the unsaturated zone;
- infiltration rate is uniform throughout the unsaturated zone;
- decay of the contaminant source is not considered (*i.e.*, infinite source mass);
- contaminant is not present as a free-phase product;
- maximum possible concentration in the leachate is equivalent to the solubility limit of the chemical in water under the defined site conditions;

- the groundwater aquifer is unconfined;
- groundwater flow is uniform and steady;
- co-solubility and oxidation/reduction effects are not considered;
- attenuation of the contaminant in the saturated zone is assumed to be one dimensional with respect to sorption-desorption, dispersion, and biological degradation;
- dispersion in groundwater is assumed to occur in the longitudinal and transverse directions only and diffusion is not considered;
- mixing of the leachate with the groundwater is assumed to occur through mixing of leachate and groundwater mass fluxes; and
- dilution of the plume by groundwater recharge down-gradient of the source is not considered.

12.1.2 Guideline Calculation

The soil remediation guideline protective of groundwater uses is calculated in the same way for both groundwater uses noted at the start of this section, using the corresponding surface water quality guideline (Table 8) as the starting point for each. However, as noted above, the lateral offset between the point at which the contaminated soil is measured and the surface water body (parameter "x" in the equation for DF4 below) is assumed to be 10 m for aquatic life, and 0 m for human drinking water.

The model considers four processes:

1. partitioning from soil to leachate;
2. transport of leachate from base of contamination to water table;
3. mixing of leachate and groundwater; and,
4. groundwater transport down-gradient to a discharge point.

For each of these four processes, a dilution factor was calculated (DF1 through DF4, respectively). DF1 has units of (mg/kg)/(mg/L) or L/kg. The other three dilution factors are dimensionless [units of (mg/L)/(mg/L)]. The overall dilution factor is used to calculate the soil concentration that is protective of groundwater using the following equations:

$$SQG_{GR} = SWQG \times DF$$

$$DF = DF1 \times DF2 \times DF3 \times DF4$$

where: SQG_{GR}= soil remediation guideline protective of groundwater pathways (mg/kg)

SWQG= corresponding surface water quality guideline (drinking water or aquatic life) (mg/L)
DF = overall dilution factor (L/kg)
DF1 = dilution factor for process 1 (L/kg)
DF2 = dilution factor for process 2 (dimensionless)
DF3 = dilution factor for process 3 (dimensionless)
DF4 = dilution factor for process 4 (dimensionless)

Dilution Factor 1

Dilution factor 1 (DF1) is the ratio of the concentration of a contaminant in soil to the concentration in leachate that is in contact with the soil. This "dilution factor" represents the three phase partitioning between contaminant sorbed to soil, contaminant dissolved in pore water (*i.e.*, as leachate), and contaminant present as soil vapour. DF1 is calculated using the following equation:

$$DF1 = K_d + \frac{(\theta_w + H' \times \theta_a)}{\rho_b}$$

where:

$DF1$ = dilution factor 1 (L/kg)
K_d = soil to water partition coefficient (L/kg)
θ_w = water filled porosity (dimensionless)
H' = dimensionless Henry's law constant (dimensionless)
θ_a = air filled porosity (dimensionless)
ρ_b = dry soil bulk density (g/cm^3)

Dilution Factor 2

Dilution factor 2 (DF2) is the ratio of the concentration of a contaminant in leachate that is in contact with the soil to the concentration in pore water just above the groundwater table. DF2 takes the value 1.00 (*i.e.*, no dilution) for generic guidelines because it is assumed at Tier 1 that the contaminated soil extends down to the water table. Note that DF2 can be calculated on a site-specific basis at Tier 2 (AENV, 2009b).

Dilution Factor 3

Dilution factor 3 (DF3) is the ratio of the concentration of a chemical in pore water just above the groundwater table, to the concentration in groundwater beneath the source. This dilution factor reflects a decrease in concentration as leachate mixes with uncontaminated groundwater. DF3 is a function of groundwater velocity, infiltration rate, source length, and mixing zone thickness. The mixing zone thickness is calculated as being due to two processes: i) mixing due to dispersion, and ii) mixing due to infiltration rate. The equations used are as follows:

$$DF3 = 1 + \frac{Z_d \times V}{I \times X}$$

$$Z_d = r + s$$

$$r = 0.01 \times X$$

$$s = d_a \left\{ 1 - exp\left(\frac{-2.178 \times X \times I}{V \times d_a} \right) \right\}$$

$$V = K \times i$$

where:

DF3 = dilution factor 3 (dimensionless)
Z_d = average thickness of mixing zone (m)
V = Darcy velocity in groundwater (m/year)
I = infiltration rate (m/year)
X = length of contaminated soil (m)
r = mixing depth due to dispersion (m)
s = mixing depth due to infiltration rate (m)
d_a = unconfined aquifer thickness (m)
K = aquifer hydraulic conductivity (m/year)
i = lateral hydraulic gradient in aquifer (dimensionless)

Note that the parameter Z_d takes the fixed value of 2 m for the drinking water pathway, but is calculated as above for all other pathways.

Dilution Factor 4

Dilution factor 4 (DF4) accounts for the processes of dispersion and biodegradation as groundwater travels downgradient from beneath the source of contamination, and is the ratio of the concentration of a chemical in groundwater beneath the source, to the concentration in groundwater at a distance of 10 m (at Tier 1 for aquatic life) downgradient of the source. Consistent with AENV (2009a), the time independent version of the equation to calculate DF4 was used:

$$DF4 = \frac{2}{\exp(A) \times [erf(C) - erf(D)]}$$

$$A = \frac{x}{2D_x}\left\{1 - \left(1 + \frac{4L_s D_x}{v}\right)^{1/2}\right\}$$

$$C = \frac{y + Y/2}{2(D_y x)^{1/2}}$$

$$D = \frac{y - Y/2}{2(D_y x)^{1/2}}$$

$$L_s = \frac{0.6931}{t_{1/2s}} \times \exp(-0.07d)$$

$$v = \frac{V}{\theta_t R_s}$$

$$R_s = 1 + \frac{\rho_b K_d}{\theta_t}$$

$$D_x = 0.1x$$

$$D_y = 0.01x$$

where:

DF4 = dilution factor 4 (dimensionless)
erf = the error function
A = dimensionless group A (dimensionless)
C = dimensionless group C (dimensionless)
D = dimensionless group D (dimensionless)
x = distance to source (10 m, aquatic life and wildlife watering, 0 m other water uses)
D_x = dispersivity in the direction of groundwater flow (m)
L_s = decay constant (1/year)
v = velocity of the contaminant (m/year)
y = distance to receptor perpendicular to groundwater flow (m)
Y = source width (m)
D_y = dispersivity perpendicular to the direction of groundwater flow (m)
$t_{1/2s}$ = decay half-life of contaminant in saturated zone of aquifer (years)
d = water table depth (m)

V = Darcy velocity in groundwater (m/year)
θ_t = total soil porosity (dimensionless)
R_s = retardation factor in saturated zone (dimensionless)
ρ_b = dry soil bulk density (g/cm^3)
K_d = soil to water partition coefficient (mL/g)

Aquatic Life

Substituting appropriate values from Tables 3, 5, 6, and 8 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives values of:

- 10 mg/kg (MEA, coarse soil; Table 9);
- 300,000 mg/kg (MEA, fine soil; Table 10);
- 45 mg/kg (DEA, coarse soil; Table 11); and,
- 500,000 mg/kg (DEA, fine soil; Table 12).

Protection of Domestic Use Aquifer

Substituting appropriate values from Tables 3, 5, 6, and 8 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives values of:

- 40 mg/kg (MEA, coarse soil; Table 9);
- 20 mg/kg (MEA, fine soil; Table 10);
- 3.5 mg/kg (DEA, coarse soil; Table 11); and,
- 2.0 mg/kg (DEA, fine soil; Table 12).

12.2 Groundwater Remediation Guidelines

Groundwater remediation guidelines for groundwater pathways were calculated using the model and equations from AENV (2009a).

12.2.1 Potable Groundwater

If contaminated groundwater is considered a domestic use aquifer, there is no offset assumed between contamination and a potential future water well; therefore, the Source Guidance Value for Groundwater (0.6 mg/L, MEA; 0.06 mg/L, DEA) applies directly to groundwater (Tables 13 and 14).

12.2.2 Aquatic Life

Assumptions implicit in the model include the following:

- the soil/aquifer material in the saturated zone is physically and chemically homogeneous;

- decay of the contaminant source is not considered (*i.e.*, infinite source mass);
- the contaminant is not present as a free-phase product;
- groundwater flow is uniform and steady;
- co-solubility and oxidation/reduction effects are not considered;
- dispersion is assumed to occur in the longitudinal and transverse directions only and diffusion is not considered; and,
- dilution of the plume by groundwater recharge down-gradient of the source is not considered.

Guideline Calculation

The groundwater remediation guideline protective of aquatic life is calculated using the following equations.

$$GWQG_{GR} = SWQG \times DF4$$

where:

$GWQG_{GR}$= groundwater quality guideline protective of aquatic life (mg/L)
$SWQG_{FL}$= surface water quality guideline protective of aquatic life (mg/L)
DF4 = dilution factor for process 4 (L/kg)

Dilution factor 4 is calculated in the same way as described in Section 12.1.2

Substituting appropriate values from Tables 3, 5, 6, and 8 into this equation and rounding to 1 significant figure with a 5 or 0 in the second figure gives values of:

- 1 mg/L (MEA, coarse soil; Table 13);
- 30,000 mg/L (MEA, fine soil; Table 13);
- 5 mg/L (DEA, coarse soil; Table 14); and,
- 65,000 mg/L (DEA, fine soil; Table 14).

13. GUIDELINE APPLICATION

The soil and groundwater guidelines calculated in this report (Tables 9 to 14) can be applied as specified in AENV (2009a) as Tier 1 guidelines, and can be used as the basis to develop Tier 2 guidelines as indicated in AENV (2009b). However, care must be taken to ensure that the analytical data with which these guidelines are compared was collected using an appropriate method.

Application of the guidelines in this document is only valid when compared to analytical data that were obtained using a method that is able to achieve quantitative and repeatable recovery of alkanolamines from a soil matrix similar to soils at the site in question. The method presented in Appendix C is recommended for analyzing alkanolamines in Alberta. Alternative methods are acceptable, but must meet or exceed the performance criteria in Appendix C.

14. REFERENCES

(See also References in Appendices A and B)

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TABLES

Table 1. Common Synonyms and Trade Names for the Amines

Monoethanolamine	Diethanolamine
MEA	DEA
ethylolamine	diethylolamine
2-aminoethanol	2,2'-aminodiethanol
b-aminoethylalcohol	2,2'-iminodiethanol
ethanolamine	bis(2-hydroxyethyl)amine
b-hydroxyethylamine	2,2'-dihydroxydiethylamine
colamine	b,b'-hydroxydiethylamine
	2,2'-iminobis-ethanol

Table 2. Physical and Chemical Properties for the Amines

Property	Units	MEA	DEA	Source
Formula	-	C_2H_7NO	$C_4H_{11}NO_2$	1
CAS number	-	141-43-5	111-42-2	1
Molecular weight	g/mole	61.09	105.14	2
Conversion factor (1 mg/m^3 =)	ppm	0.39	0.23	4
pH (10% solution at 25 °C)	-	12.0	11.5	3
Acid dissociation constant (pK_a)	-	9.68	9.01	3
Melting point (at 760 torr)	°C	10	28	2
Boiling point (at 760 torr)	°C	171	268	2
Specific gravity (at 25/4 °C)	g/cm^3	1.011	1.088	2
Vapour pressure (at 20 °C)	Pa	53	<1.3	4,7
Solubility (at 25 °C)	mg/L	miscible	miscible	2
Henry's law constant	atm·m^3/mol	2.5×10^{-7}	5.4×10^{-14}	5
Dimensionless Henry's law constant	-	1.7×10^{-6}	2.2×10^{-12}	2
Soil-water partition coefficient (K_d)	L/kg	2.21-4.91	1.9-6.4	8
Organic carbon partition coefficient (K_{oc})	log	-0.223	-0.308	6
n-Octanol-water partition coefficient (K_{ow})	log	-1.31	-1.43	4,6
Bioconcentration factor (BCF)	log	-1.23	-1.32	5

Sources:

[1] CRC (1996)

[2] Dow Chemical Company (1988)

[3] Lewis (1992)

[4] Verschueren (1983)

[5] Calculated from Lyman et al. (1982) as reported in Davis and Carpenter (1997)

[6] Calculated from K_{ow} using Gerstl and Kliger (1990) equation provided in Boethling and Mackay (2000; Table 8.1)

[7] Recalculated from Verschueren (1983) using the conversion 1 mm Hg = 1 torr = 133 Pa

[8] Sorensen et al. (1997,1998)

Table 3. Chemical-Specific Parameter Values for MEA and DEA

Parameter	Unit	MEA	DEA	Rationale
Human Toxicity				
Tolerable Daily Intake (oral exposure)	mg/kg-bw/day	0.05	0.005	see Section 6.9
Tolerable Concentration (inhalation exposure)	mg/m^3	na	na	negligible vapour pressure
Human Background Exposure				
Estimated daily intake	mg/kg-bw/day	0	0	see Section 2.5
Ambient air concentration	mg/m^3	0	0	see Section 2.5
Background soil concentration	mg/kg	0	0	see Section 2.5
Soil allocation factor	-	0.25	0.25	see Section 8.1
Water allocation factor	-	0.25	0.25	see Section 8.1
Human Adsorption				
Adsorption factor - gut	-	1.0	1.0	assumed
Adsorption factor - gut	-	1.0	1.0	assumed
Adsorption factor - gut	-	1.0	1.0	assumed
Chemical and Physical Properties				
Soil Organic Carbon/Water Partition Coefficient (Koc)	L/kg	0.598	0.492	see Table 2
Soil/Porewater Partition Coefficient (Kd)	L/kg	2.21	1.9	see Section 3.1
Dimensionless Henry's law coeffcient	(mg/L)/(mg/L)	1.7×10^{-6}	2.2×10^{-12}	see Table 2
Dynamic viscosity of vapour	g/cm.s	1.73×10^{-4}	1.73×10^{-4}	AENV (2009a)
Diffusion coefficient in air	cm^2/s	na	na	negligible vapour pressure
Degradation				
Degradation half life (saturated)	days	275	275	see Section 3.4.3

Notes:
na = not applicable

Table 4. Human Receptor Characteristics

Parameter	Symbol	Unit	Toddler	Adult
Body Weight	BW	kg	16.5	70.7
Air Inhalation Rate	IR	m^3/d	9.3	15.8
Soil (Dust) Inhalation Rate	IR_S	kg/d	7.1×10^{-9}	1.2×10^{-8}
Water Ingestion Rate	WIR	L/d	0.6	1.5
Soil Ingestion Rate	SIR	kg/d	0.00008	0.00002
Skin Surface Area				
- Hands	SA_H	m^2	0.043	0.089
- Other	SA_O	m^2	0.258	0.25
Dermal Loading to Skin				
- Hands	DL_H	kg/m^2-event	0.001	0.001
- Other	DL_O	kg/m^2-event	0.0001	0.0001
Dermal Exposure Frequency	EF	events/d	1	1
Exposure Term, agricultural and residential/parkland	ET	-	1	1
Exposure Term, commercial and industrial	ET	-	0.2747	0.2747
Exposure Term, agricultural and residential/parkland	ET_1	-	1	1
Exposure Term, commercial and industrial	ET_1	-	0.6593	0.6593
Exposure Term, agricultural and residential/parkland	ET_2	-	1	1
Exposure Term, commercial and industrial	ET_2	-	0.4167	0.4167

Notes:

All parameter values from AENV (2009a)

Table 5. Soil and Hydrogeological Parameters

Parameter	Symbol	Unit	Fine Soil	Coarse Soil
Soil Bulk Density	ρ_B	*kg/L*	1.4	1.7
Soil Total Porosity	θ_t	*cm³/cm³*	0.47	0.36
Soil Moisture-Filled Porosity	θ_w	*cm³/cm³*	0.168	0.119
Soil Vapour-Filled Porosity	θ_a	*cm³/cm³*	0.302	0.241
Soil Vapour-Filled Porosity in Floor Cracks	θ_a	*cm³/cm³*	0.47	0.36
Gravimetric Water Content	MC	*g/g*	0.12	0.07
Fraction of Organic Carbon	f_{oc}	*mass/mass*	0.005	0.005
Saturated Hydraulic Conductivity	K	*m/y*	32	320
Hydraulic Gradient	i	*m/m*	0.028	0.028
Recharge (Infiltration) Rate	I	*m/y*	0.012	0.06
Soil Permeability to Vapour Flow	k_v	*cm²*	10^{-9}	$6x10^{-8}$

Notes:

All parameter values from AENV (2009a)

Table 6. Site Characteristics

Parameter	Symbol	Unit	Value
Contaminant Source Width	Y	m	10
Contaminant Source Length	X	m	10
Contaminant Source Depth	Z	m	3
Distance to Surface Water	x	m	10
Distance to Potable Water User	x	m	0
Distance to Agricultural Water User	x	m	0
Distance from Contamination to Building Slab	L_T	cm	30
Depth to Groundwater (water table)	d	m	3
Depth of unconfined aquifer	d_a	m	5
Mixing zone - potable water pathway only	b	m	2

Notes:

All parameter values from AENV (2009a)

Table 7. Building Parameters

Parameter	Symbol	Unit	Residential Basement	Residential Slab-on-Grade	Commercial Slab-on-Grade
Building Length	L_B	*cm*	1,225	1,225	2,000
Building Width	W_B	*cm*	1,225	1,225	1,500
Building Height (including basement)	H_B	*cm*	360	360	300
Area of Substructure	A_B	*cm*^{2}	2.7×10^6	1.5×10^6	3.0×10^6
Thickness of Floor Slab	L_{crack}	*cm*	11.25	11.25	11.25
Depth of Floor Slab Below Ground	Z_{crack}	*cm*	244	11.25	11.25
Distance from Source to Slab:	L_T	*cm*			
surface soil			30	30	30
subsoil			30	139	139
Crack Area	A_{crack}	*cm*^{2}	994.5	994.5	1,846
Crack Length	X_{crack}	*cm*	4,900	4,900	7,000
Air Exchange Rate	ACH	*exch/hr*	0.5	0.5	0.9
Pressure Differential	ΔP	*g/cm.s*^{2}	40	40	20

Notes:

All parameter values from AENV (2009a)

Table 8. Surface Water Quality Guidelines for MEA and DEA

	MEA	DEA
Water Use	**(mg/L)**	**(mg/L)**
Human drinking water ("Source Guidance Value for Groundwater")	0.6	0.06
Freshwater aquatic life	0.075	0.45
Irrigation [1]	n/c	n/c
Livestock watering [2]	n/c	n/c
Wildlife watering [3]	n/c	n/c

Notes:

n/c = not calculated

1. *guideline protective of irrigation not calculated due to lack of toxicity data relevant to irrigation.*
2. *guideline not calculated due to the lack of toxicity information for livestock species.*
3. *guideline not calculated due to the lack of toxicity information for wildlife species.*

Table 9. Soil Remediation Guidelines for MEA - Coarse Soil

	Guideline Value (mg/kg)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Overall Guideline	**10**	**10**	**10**	**10**	**10**
Human Exposure Pathways					
Direct soil contact	n/a	1,500	1,500	2,000	10,000
Vapour inhalation	n/a	n/c	n/c	n/c	n/c
Protection of domestic use aquifer	40	40	40	40	40
Produce, milk and meat check [1]	n/c	n/c	n/c	n/c	n/c
Off-site migration [2]	n/a	n/a	n/a	20,000	20,000
Ecological Exposure Pathways					
Direct soil contact	1,500	1,500	1,500	1,500	1,500
Nutrient and Energy cycling check [3]	n/c	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [4]	n/c	n/c	n/c	n/c	n/c
Protection of freshwater aquatic life	10	10	10	10	10
Off-site migration [2]	n/a	n/a	n/a	20,000	20,000

Notes:

See Section 13 for Analytical Method Requirements

n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

1. *Produce, meat and milk check not calculated - amines not expected to accumulate in produce, milk, or meat.*
2. *Offsite migration not considered a concern given the degradability of amines in conditions likely to be found at surface (Section 3.4.3).*
3. *Nutrient and energy cycling check not calculated - insufficient data*
4. *Livestock soil and food ingestion not expected to be a concern, amines expected to be lost rapidly from surface soil, and not accumulate into fodder.*

Table 10. Soil Remediation Guidelines for MEA - Fine Soil

	Guideline Value (mg/kg)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Overall Guideline	**20**	**20**	**20**	**20**	**20**
Human Exposure Pathways					
Direct soil contact	n/a	1,500	1,500	2,000	10,000
Vapour inhalation	n/a	n/c	n/c	n/c	n/c
Protection of domestic use aquifer	20	20	20	20	20
Produce, milk and meat check [1]	n/c	n/c	n/c	n/c	n/c
Off-site migration [2]	n/a	n/a	n/a	20,000	20,000
Ecological Exposure Pathways					
Direct soil contact	1,500	1,500	1,500	1,500	1,500
Nutrient and Energy cycling check [3]	n/c	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [4]	n/c	n/c	n/c	n/c	n/c
Protection of freshwater aquatic life	300,000	300,000	300,000	300,000	300,000
Off-site migration [2]	n/a	n/a	n/a	20,000	20,000

Notes:

See Section 13 for Analytical Method Requirements

n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

1. *Produce, meat and milk check not calculated - amines not expected to accumulate in produce, milk, or meat.*
2. *Offsite migration not considered a concern given the degradability of amines in conditions likely to be found at surface (Section 3.4.3).*
3. *Nutrient and energy cycling check not calculated - insufficient data*
4. *Livestock soil and food ingestion not expected to be a concern, amines expected to be lost rapidly from surface soil, and not accumulate into fodder.*

Table 11. Soil Remediation Guidelines for DEA - Coarse Soil

	Guideline Value (mg/kg)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Overall Guideline	**3.5**	**3.5**	**3.5**	**3.5**	**3.5**
Human Exposure Pathways					
Direct soil contact	n/a	150	150	200	1,000
Vapour inhalation	n/a	n/c	n/c	n/c	n/c
Protection of domestic use aquifer	3.5	3.5	3.5	3.5	3.5
Produce, milk and meat check [1]	n/c	n/c	n/c	n/c	n/c
Off-site migration [2]	n/a	n/a	n/a	2,000	2,000
Ecological Exposure Pathways					
Direct soil contact	1,000	1,000	1,000	2,000	2,000
Nutrient and Energy cycling check [3]	n/c	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [4]	n/c	n/c	n/c	n/c	n/c
Protection of freshwater aquatic life	45	45	45	45	45
Off-site migration [2]	n/a	n/a	n/a	15,000	15,000

Notes:

See Section 13 for Analytical Method Requirements

n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

1. *Produce, meat and milk check not calculated - amines not expected to accumulate in produce, milk, or meat.*
2. *Offsite migration not considered a concern given the degradability of amines in conditions likely to be found at surface (Section 3.4.3).*
3. *Nutrient and energy cycling check not calculated - insufficient data*
4. *Livestock soil and food ingestion not expected to be a concern, amines expected to be lost rapidly from surface soil, and not accumulate into fodder.*

Table 12. Soil Remediation Guidelines for DEA - Fine Soil

	Guideline Value (mg/kg)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Overall Guideline	**2.0**	**2.0**	**2.0**	**2.0**	**2.0**
Human Exposure Pathways					
Direct soil contact	n/a	150	150	200	1,000
Vapour inhalation	n/a	n/c	n/c	n/c	n/c
Protection of domestic use aquifer	2.0	2.0	2.0	2.0	2.0
Produce, milk and meat check [1]	n/c	n/c	n/c	n/c	n/c
Off-site migration [2]	n/a	n/a	n/a	2,000	2,000
Ecological Exposure Pathways					
Direct soil contact	1,000	1,000	1,000	2,000	2,000
Nutrient and Energy cycling check [3]	n/c	n/c	n/c	n/c	n/c
Livestock soil and food ingestion [4]	n/c	n/c	n/c	n/c	n/c
Protection of freshwater aquatic life	500,000	500,000	500,000	500,000	500,000
Off-site migration [2]	n/a	n/a	n/a	15,000	15,000

Notes:

See Section 13 for Analytical Method Requirements

n/a = exposure pathway not applicable in this scenario.

n/c = not calculated

1. *Produce, meat and milk check not calculated - amines not expected to accumulate in produce, milk, or meat.*
2. *Offsite migration not considered a concern given the degradability of amines in conditions likely to be found at surface (Section 3.4.3).*
3. *Nutrient and energy cycling check not calculated - insufficient data*
4. *Livestock soil and food ingestion not expected to be a concern, amines expected to be lost rapidly from surface soil, and not accumulate into fodder.*

Table 13. Groundwater Remediation Guidelines for MEA

	Guideline Value (mg/L)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Lowest Guideline (Coarse)	**0.6**	**0.6**	**0.6**	**0.6**	**0.6**
Lowest Guideline (Fine)	**0.6**	**0.6**	**0.6**	**0.6**	**0.6**
Water Use					
Potable groundwater	0.6	0.6	0.6	0.6	0.6
Vapour inhalation from groundwater [1]					
Coarse soil	n/a	n/c	n/c	n/c	n/c
Fine soil	n/a	n/c	n/c	n/c	n/c
Groundwater protective of eco-contact [2]					
Coarse soil	n/c	n/c	n/c	n/c	n/c
Fine soil	n/c	n/c	n/c	n/c	n/c
Groundwater protective of freshwater aquatic life					
Coarse soil	1	1	1	1	1
Fine soil	30,000	30,000	30,000	30,000	30,000
Groundwater used for irrigation [3]	n/c	n/c	n/c	n/c	n/c
Groundwater used for livestock watering [4]	n/c	n/c	n/c	n/c	n/c
Groundwater used for wildlife watering [5]	n/c	n/c	n/c	n/c	n/c

Notes:

n/a = water use not applicable in this scenario.

n/c = not calculated

1. *Pathway not a concern - amines have negligible vapour pressure*
2. *See section 11.1.2*
3. *Groundwater protective of irrigation not calculated due to lack of toxicity data relevant to irrigation.*
4. *Livestock watering groundwater guideline not calculated due to the lack of toxicity information for livestock species.*
5. *Wildlife watering groundwater guideline not calculated due to the lack of toxicity information for wildlife species.*

Table 14. Groundwater Remediation Guidelines for DEA

	Guideline Value (mg/L)				
Land Use:	**Natural Area**	**Agricultural**	**Residential**	**Commercial**	**Industrial**
Lowest Guideline (Coarse)	**0.06**	**0.06**	**0.06**	**0.06**	**0.06**
Lowest Guideline (Fine)	**0.06**	**0.06**	**0.06**	**0.06**	**0.06**
Water Use					
Potable groundwater	0.06	0.06	0.06	0.06	0.06
Vapour inhalation from groundwater [1]					
Coarse soil	n/a	n/c	n/c	n/c	n/c
Fine soil	n/a	n/c	n/c	n/c	n/c
Groundwater protective of eco-contact [2]					
Coarse soil	n/c	n/c	n/c	n/c	n/c
Fine soil	n/c	n/c	n/c	n/c	n/c
Groundwater protective of freshwater aquatic life					
Coarse soil	5	5	5	5	5
Fine soil	65,000	65,000	65,000	65,000	65,000
Groundwater used for irrigation [3]	n/c	n/c	n/c	n/c	n/c
Groundwater used for livestock watering [4]	n/c	n/c	n/c	n/c	n/c
Groundwater used for wildlife watering [5]	n/c	n/c	n/c	n/c	n/c

Notes:

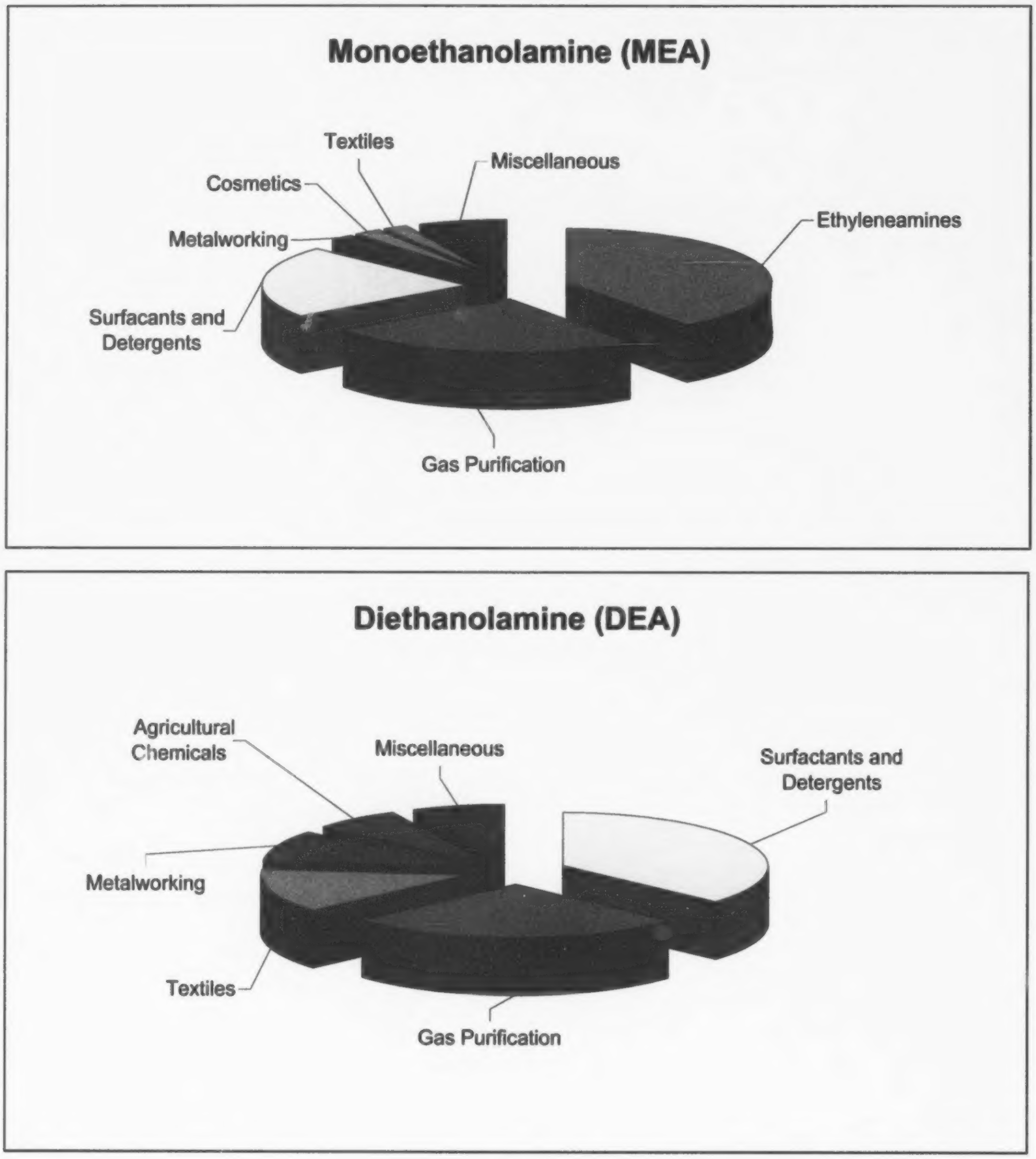
n/a = water use not applicable in this scenario.

n/c = not calculated

1. *Pathway not a concern - amines have negligible vapour pressure*
2. *See section 11.1.2*
3. *Groundwater protective of irrigation not calculated due to lack of toxicity data relevant to irrigation.*
4. *Livestock watering groundwater guideline not calculated due to the lack of toxicity information for livestock species.*
5. *Wildlife watering groundwater guideline not calculated due to the lack of toxicity information for wildlife species.*

FIGURES

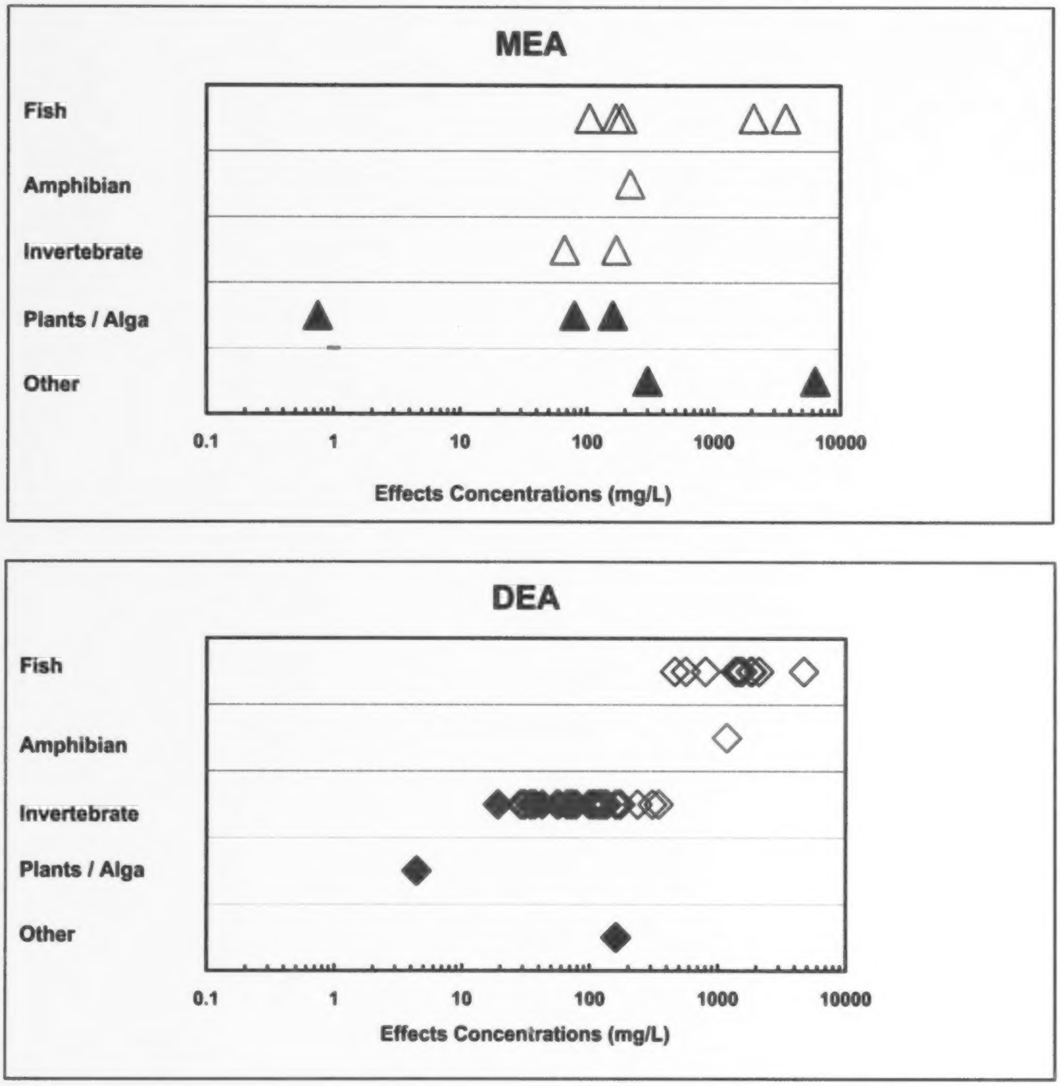
Figure 1. Major Uses of MEA and DEA



Monoethanolamine (MEA)

Diethanolamine (DEA)

Figure 2. Effects Concentrations of MEA and DEA to Freshwater Aquatic Organisms



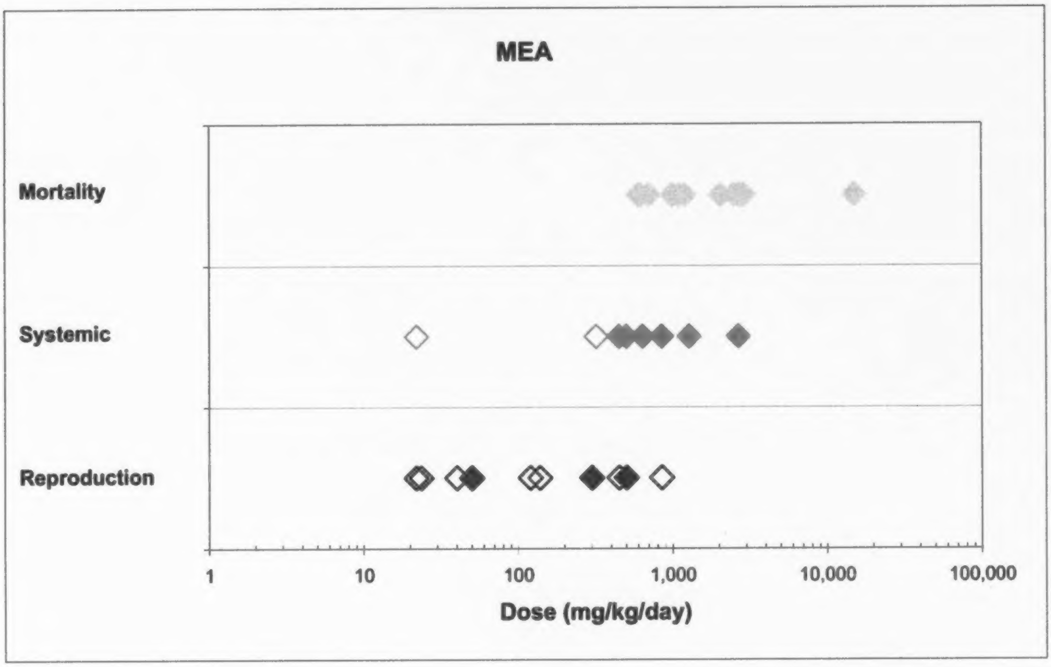
Notes:

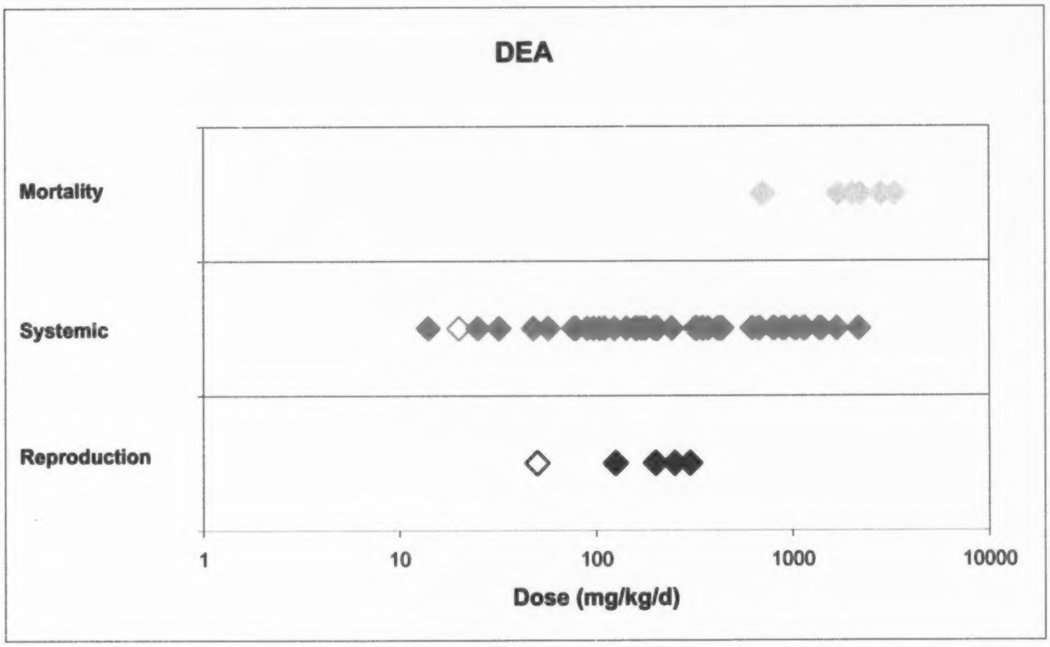
Only primary and secondary data presented

Solid symbol = chronic

Hollow symbol = acute

Figure 3. Oral Toxicity of MEA and DEA to Mammalian Species





Notes:
Solid Symbol = Effects
Hollow Symbol = No Effect

APPENDIX A

MEA
DEGRADATION AND TOXICITY DATA

Table A-1 Summary of Available Data on MEA Biodegradation
Table A-2 Toxicity of MEA to Freshwater Aquatic Life
Table A-3 Toxicity of MEA to Marine Aquatic Life
Table A-4 Toxicity of MEA to Terrestrial Plants
Table A-5 Toxicity of MEA to Terrestrial Invertebrates
Table A-6 Toxicity of MEA to Mammalian Species

Table A-1. Summary of Available Data on MEA Biodegradation

Test Method	Test Duration	Aerobic/ Anaerobic	Initial Compound Concentration	% Removed	Inoculum or Medium	Interpreted Half Life	Rates / Comments	Reference
Studies with Relevance to Subsurface Conditions								
batch cultures; IEC analysis	64 days	aerobic	31,000 mg/kg	15%	contaminated soil groundwater slurry	225 days	Complete degradation achieved after phosphate amendment. Interpreted half life is based on 64 days of unamended degradation	Mrklas et al. (2004)
batch cultures; IEC analysis	98 days	anaerobic	31,000 mg/kg	20%	contaminated soil groundwater slurry	275 days	Anaerobic degradation rate interpreted as zero order. Half life was the extraolated time to drop to half o finitial concentration.	Mrklas et al. (2004)
batch cultures; IC analysis	22 days	aerobic	500 mg/kg	100%	soil at 30% of field capacity	13.5 days	linear kinetics	Sorensen et al. (1997)
batch cultures; IC analysis	82 days	anaerobic	500 mg/kg	52%	soil at 30% of field capacity	80 days	linear kinetics	Sorensen et al. (1997)
batch cultures; IC analysis	29 days	aerobic	1,533 mg/kg	100%	soil at ambient moisture	6 days	ambient (lab) temperature	Ndegwa et al. (2004)
batch cultures; IC analysis	29 days	anaerobic	1,533 mg/kg	100%	soil at ambient moisture	3 days	ambient (lab) temperature	Ndegwa et al. (2004)
batch cultures; IC analysis	61 days	aerobic	267 mg/kg	100%	soil at ambient moisture	2 days	ambient (lab) temperature	Ndegwa et al. (2004)
batch cultures; IC analysis	61 days	aerobic	267 mg/kg	100%	soil at ambient moisture	7 days	cool temperature (5-10C)	Ndegwa et al. (2004)
Other Studies								
BOD Test	5 days	aerobic	nd	34%	sewage	nd	Lag period = 2 d	Younge et al. (1968)
BOD Test	20 days	aerobic	nd	40%	sewage	nd	Lag period = 2 d	Younge et al. (1968)
BOD Test	5 days	aerobic	2.5 ppm	0%	filtered sewage	nd		Lamb and Jenkins (1952)
BOD Test	10 days	aerobic	2.5 ppm	58%	filtered sewage	nd		Lamb and Jenkins (1952)
BOD Test	20 days	aerobic	2.5 ppm	64%	filtered sewage	nd		Lamb and Jenkins (1952)
BOD Test	>20 days	aerobic	2.5 ppm	75%	filtered sewage	nd		Lamb and Jenkins (1952)
BOD Test	5 days	aerobic	nd	71%	filtered sewage	nd		Bridie et al. (1979a)
BOD Test	5 days	aerobic	100 ppm	71%	filtered sewage	nd	14 % DOC removal	Urano and Kata (1986)
BOD Test	10 days	aerobic	nd	65%	sewage	nd		Mills and Stack (1952)
BOD Test	5 days	aerobic	nd	65%	sewage	nd		Alexander and Batchelder (1975)
BOD Test	10 days	aerobic	nd	65%	sewage	nd		Alexander and Batchelder (1975)
BOD Test	20 days	aerobic	nd	65%	sewage	nd		Alexander and Batchelder (1975)

Notes:

Biochemical oxygen demand (BOD) is defined as parts of oxygen consumed per part of compound during degradation.

Table A-2. Toxicity of MEA to Freshwater Aquatic Life

Scientific Name	Common Name	Group	Acute or Chronic	Endpoint	Effect	Exposure Duration (Days)	Exposure Type	Chemical Analysis	Concentration (mg/L)	Control Type	Reference	Comment
Primary Data												
Daphnia magna	Water flea	Invertebrates	Acute	LC50	Mortality	2	Static	Measured	67	Satisfactory	Vizon (2006)	Comissioned for this report
Oncorhynchus mykiss	Rainbow trout	Fish	Acute	LC50	Mortality	4	Static	Measured	105	Satisfactory	Vizon (2006)	Comissioned for this report
Hyalella azteca	Amphipod	Invertebrates	Acute	LC50	Mortality	4	Static	Measured	170	Satisfactory	Vizon (2006)	Comissioned for this report
Carassius auratus	Goldfish	Fish	Acute	LC50	Mortality	4	Static	Measured	170	Satisfactory	Bridie et al. (1979b)	
Carassius auratus	Goldfish	Fish	Acute	LC50	Mortality	4	Static	Measured	190	Satisfactory	Bridie et al. (1979b)	
Pimephales promelas	Fathead minnow	Fish	Acute	LC50	Mortality	4	Flow-through	Measured	2,070	Satisfactory	Geiger et al. (1990)	
Secondary Data												
Scenedesmus quadricauda	Green algae	Algae	Chronic	IC03	Growth	7	Static	Nominal	0.75	Satisfactory	Bringmann and Kuhn (1980)	Lowest valid datapoint
Isochrysis galbana	Alga	Algae	Chronic	EC50	Growth	4	NR	Nominal	80	Satisfactory	Roseth et al. (1996)	
Isochrysis galbana	Alga	Algae	Chronic	EC50	Growth	2	NR	Nominal	160	Satisfactory	Roseth et al. (1996)	
Xenopus laevis	African clawed frog	Amphibians	Acute	LC50	Mortality	2	Static	Nominal	220	Satisfactory	De Zwart and Slooff (1987)	
Entosiphon sulcatum	Flagellate euglenoid	Protozoa	Chronic	IC03	Growth	3	Static	Nominal	300	Satisfactory	Bringmann and Kuhn (1980)	
Danio rerio	Zebra fish	Fish	Acute	LC50	Mortality	4	NR	Nominal	3,684	Satisfactory	Groth et al (1993)	
Pseudomonas putida	Bacteria	Protozoa	Acute	IC03	Growth	0.67	Static	Nominal	6,300	Satisfactory	Bringmann and Kuhn (1980)	
Unacceptable Data												
Scenedesmus quadricauda	Green algae	Algae	Chronic	NR	Growth	8	Static	Nominal	0.97	NR	Bringmann and Kuhn (1978)	Media not specified
Daphnia magna	Water flea	Crustaceans	NV	NR	Mortality	NR	NR	Nominal	1	NR	Apostol (1975)	Endpoint not specified
Microcystis aeruginosa	Blue-green algae	Protozoa	Chronic	NR	Growth	8	NR	Nominal	1.6	NR	Bringmann (1975)	Endpoint not specified
Anacystis aeruginosa	Blue-green algae	Protozoa	Chronic	NR	Growth	8	Static	Nominal	1.6	NR	Bringmann and Kuhn (1978)	Media not specified
Microcystis aeruginosa	Blue-green algae	Protozoa	Chronic	NR	Growth	8	NR	Nominal	2.1	NR	Bringmann (1975)	Endpoint not specified
Anacystis aeruginosa	Blue-green algae	Protozoa	Chronic	NR	Growth	8	Static	Nominal	2.1	NR	Bringmann and Kuhn (1978)	Media not specified
Chlorococcales	Green algae order	Algae	Acute	EC10	Growth	1	Static	NR	31	NR	Krebs (1991)	No control or insufficient data
Semotilus atromaculatus	Creek chub	Fish	Acute	LC0	Mortality	1	Static	Nominal	40	NR	Gillette et al. (1952)	No effect at concentration tested
Daphnia magna	Water flea	Crustaceans	Acute	LC0	Mortality	1	Static	Nominal	52	NR	Bringmann and Kuhn (1977)	No effect at concentration tested
Semotilus atromaculatus	Creek chub	Fish	Acute	LC100	Mortality	1	Static	Nominal	70	NR	Gillette et al. (1952)	No control or insufficient data
Chlorococcales	Green algae order	Algae	Acute	EC50	Growth	1	Static	NR	70	NR	Krebs (1991)	No control or insufficient data
Lepomis macrochirus	Bluegill	Fish	Acute	LC0*	Mortality	4	Static	Nominal	75	Satisfactory	Buzzell et al. (1968)	No effect at concentration tested
Daphnia magna	Water flea	Crustaceans	NV	NR	Mortality	NR	NR	Nominal	100	NR	Apostol (1975)	Endpoint not specified
Daphnia magna	Water flea	Crustaceans	Acute	LC50	Mortality	1	Static	Nominal	140	NR	Bringmann and Kuhn (1977)	No control or insufficient data
Oncorhynchus mykiss	Rainbow trout	Fish	Acute	LC50	Mortality	1	Static	NR	150	NR	Mayer and Ellersieck (1986)	No control or insufficient data
Oncorhynchus mykiss	Rainbow trout	Fish	Acute	LC50	Mortality	4	Static	NR	150	NR	Mayer and Ellersieck (1986)	No control or insufficient data
Daphnia magna	Water flea	Crustaceans	Acute	LC100	Mortality	1	Static	Nominal	250	NR	Bringmann and Kuhn (1977)	No control or insufficient data
Entosiphon sulcatum	Flagellate euglenoid	Protozoa	NV	NR	Growth	NR	NR	NR	300	NR	Bringmann and Kuhn (1979)	Endpoint not specified
Entosiphon sulcatum	Flagellate euglenoid	Protozoa	NV	NR	Growth	NR	NR	NR	300	NR	Bringmann and Kuhn (1981)	Endpoint not specified
Lepomis macrochirus	Bluegill	Fish	Acute	LC50	Mortality	1	Static	NR	300	NR	Mayer and Ellersieck (1986)	No control or insufficient data
Lepomis macrochirus	Bluegill	Fish	Acute	LC50	Mortality	4	Static	NR	300	NR	Mayer and Ellersieck (1986)	No control or insufficient data
Lepomis macrochirus	Bluegill	Fish	Acute	LC50	Mortality	4	Static	Nominal	329	Satisfactory	Wolverton et al. (1970)	Media not specified
Gambusia affinis	Western mosquitofish	Fish	Acute	LC50	Mortality	4	Static	Nominal	338	Satisfactory	Wolverton et al. (1970)	Media not specified
Lepomis macrochirus	Bluegill	Fish	Acute	LC50	Mortality	3	Static	Nominal	346	Satisfactory	Wolverton et al. (1970)	Media not specified
Gambusia affinis	Western mosquitofish	Fish	Acute	LC50	Mortality	3	Static	Nominal	350	Satisfactory	Wolverton et al. (1970)	Media not specified

Table A-2. Toxicity of MEA to Freshwater Aquatic Life

Scientific Name	Common Name	Group	Acute or Chronic	Endpoint	Effect	Exposure Duration (Days)	Exposure Type	Chemical Analysis	Concentration (mg/L)	Control Type	Reference	Comment
Gambusia affinis	Western mosquitofish	Fish	Acute	LC50	Mortality	2	Static	Nominal	360	Satisfactory	Wolverton et al. (1970)	Media not specified
Lepomis macrochirus	Bluegill	Fish	Acute	LC50	Mortality	2	Static	Nominal	366	Satisfactory	Wolverton et al. (1970)	Media not specified
Gambusia affinis	Western mosquitofish	Fish	Acute	LC50	Mortality	1	Static	Nominal	375	Satisfactory	Wolverton et al. (1970)	Media not specified
Chilomonas paramecium	Cryptomonad	Protozoa	NV	NR	Growth	NR	NR	NR	733	NR	Bringmann and Kuhn (1981)	Endpoint not specified
Chilomonas paramecium	Cryptomonad	Protozoa	Chronic	NR	Growth	2	NR	Nominal	733	NR	Bringmann et al. (1980)	Media not specified
Danio rerio	Zebra danio	Fish	Acute	NOEC	Growth	4	NR	Nominal	1,222	Satisfactory	Groth et ai (1993)	No effect at concentration tested
Uronema parduczi	Ciliate	Invertebrates	Acute	NR	Growth	0.83	NR	NR	1,720	NR	Bringmann and Kuhn (1980)	Endpoint not specified
Uronema parduczi	Ciliate	Invertebrates	Acute	NR	Growth	0.83	NR	NR	2,945	NR	Bringmann and Kuhn (1980)	Endpoint not specified
Uronema parduczi	Ciliate	Invertebrates	NV	NR	Growth	NR	NR	NR	2,945	NR	Bringmann and Kuhn (1981)	Endpoint not specified
Oncorhynchus mykiss	Rainbow trout	Fish	Acute	LC50	Mortality	1	Flow-through	NR	>200	NR	Mayer and Ellersieck (1986)	No effect at concentration tested
Oncorhynchus mykiss	Rainbow trout	Fish	Acute	LC50	Mortality	4	Flow-through	NR	>200	NR	Mayer and Ellersieck (1986)	No effect at concentration tested
Lepomis macrochirus	Bluegill	Fish	Acute	LC50	Mortality	1	Static	Nominal	>375	Satisfactory	Wolverton et al. (1970)	No effect at concentration tested
Carassius auratus	Goldfish	Fish	Acute	LC50	Mortality	1	Static	Measured	>5,000	Satisfactory	Bridie et al. (1979b)	No effect at concentration tested
Danio rerio	Zebra fish	Fish	Acute	LC50	Mortality	1	Renewal	Nominal	>5,000	NR	Roseth et al. (1996)	No effect at concentration tested

Notes:

NV = not verified or no value

NR= not recorded

Table A-3. Toxicity of MEA to Marine Aquatic Life

Scientific Name	Common Name	Species Group	Acute or Chronic	Endpoint	Effect	Exposure Duration (days)	Exposure Type	Chemical Analysis	Concentration (mg/L)	Control Type	Reference
Unacceptable Data											
Crangon crangon	Common shrimp, sand shrimp	Invertebrate	Acute	EC50	Mortality	48 h	Renewal	Nominal	>100	Indeterminate	Portmann and Wilson (1971)

Notes:
NV = not verified or no value
NR= not recorded

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APPENDIX B

DEA
DEGRADATION AND TOXICITY DATA

Table B-1 Summary of Available Data on DEA Biodegradation
Table B-2 Toxicity of DEA to Freshwater Aquatic Life
Table B-3 Toxicity of DEA to Marine Aquatic Life
Table B-4 Toxicity of DEA to Terrestrial Plants
Table B-5 Toxicity of DEA to Terrestrial Invertebrates
Table B-6 Toxicity of DEA to Mammalian Species

Table B-1. Summary of Available Data on DEA Biodegradation

Test Method	Test Duration	Aerobic/ Anaerobic	Initial Compound Concentration	% Removed	Inoculum or Medium	Interpreted Half Life	Rates / Comments	Reference
Studies with Relevance to Subsurface Conditions								
Study of nitrate-reducing conditions	100 days	anaerobic	525 mg/L	70%	Bacteria isolated from anaerobic river sediment	29 days	70% removal was achieved in 40 days under nitrate reducing conditions.	Knapp et al. (1996)
Other Studies								
Die-away test	5 days	aerobic	105 mg/L	100%	River water	2.5 days	Spectrophotometric analysis	Emtiazi and Knapp (1994)
Die-away test	4 days	aerobic	21 ppm	5%	River water and *Pseudomonas sp.*	nd	based on mineralization to CO_2	Boethling and Alexander (1979)
Die-away test	4 days	aerobic	210 ppb	55%	River water and *Pseudomonas sp.*	nd	based on mineralization to CO_2	Boethling and Alexander (1979)
Die-away test	4 days	aerobic	21 ppt	32%	River water and *Pseudomonas sp.*	nd	based on mineralization to CO_2	Boethling and Alexander (1979)
Die-away test	14 days	aerobic	1 ppm	31%	Lake water	nd	based on mineralization to CO_2	Yordy and Alexander (1981)
Die-away test	14 days	aerobic	1 ppm	1.2%	Lake water (acid)	nd	based on mineralization to CO_2 (acid lake)	Yordy and Alexander (1981)
Die-away test	20 days	aerobic	1 ppm	53%	sewage + lake water (acid)	nd	based on mineralization to CO_2 (acid lake)	Yordy and Alexander (1981)
Inherent biodegradability test	5 days	aerobic	nd	77%	see comments	nd	Effluent from biological waste treatment plant used as inoculum (acclimated)	Bridie et al. (1979a)
Inherent biodegradability test	5 days	aerobic	various	97%	see comments	nd	Bacteria isolated from cutting fluid and a sewage population. Maximum oxidation at 500 ppm. Inhibition at 2000 mg/L	Gannon et al. (1978)
Simulation Test	7 days	aerobic	12 ppm DOC	94%	2500 ppm MLSS	nd	based on DOC removal	Gerike and Fisher (1979)
Simulation Test	7 days	aerobic	50 ppm	90%	river water and sewage	7d	based on BOD	Mills and Stack (1954)
BOD Test	5 days	aerobic	2.5 ppm	0.9%	filtered sewage	nd		Lamb and Jenkins (1952)
BOD Test	10 days	aerobic	2.5 ppm	1.4%	filtered sewage	nd		Lamb and Jenkins (1952)
BOD Test	20 days	aerobic	2.5 ppm	6.8%	filtered sewage	nd		Lamb and Jenkins (1952)
BOD Test	5 days	aerobic	nd	2%	filtered sewage	nd		Bridie et al. (1979a)
BOD Test	5 days	aerobic	nd	17	sewage	nd		Price et al. (1974)
BOD Test	10 days	aerobic	nd	72	sewage	nd		Price et al. (1974)

Table B-1. Summary of Available Data on DEA Biodegradation

Test Method	Test Duration	Aerobic/ Anaerobic	Initial Compound Concentration	% Removed	Inoculum or Medium	Interpreted Half Life	Rates / Comments	Reference
BOD Test	20 days	aerobic	nd	88	sewage	nd		Price et al. (1974)
BOD Test	5 days	aerobic	nd	2	sewage	nd	Synthetic seawater matrix	Price et al. (1974)
BOD Test	10 days	aerobic	nd	60	sewage	nd	Synthetic seawater matrix	Price et al. (1974)
BOD Test	20 days	aerobic	nd	76	sewage	nd	Synthetic seawater matrix	Price et al. (1974)
BOD Test	10 days	aerobic	100 - 1,000 ppm	0%	sewage	nd		Mills and Stack (1952)
BOD Test	10 days	aerobic	100 - 1,000 ppm	0%	sewage	nd		Mills and Stack (1952)
BOD Test	>20 days	aerobic	2 ppm	10	sewage	nd		Gerike and Fisher (1979)
BOD Test	>20 days	aerobic	2 ppm	94	enriched sewage	nd		Gerike and Fisher (1979)
BOD Test	5 days	aerobic	nd	11%	sewage	nd		Alexander and Batchelder (1975)
BOD Test	10 days	aerobic	nd	36%	sewage	nd		Alexander and Batchelder (1975)
BOD Test	20 days	aerobic	nd	58%	sewage	nd		Alexander and Batchelder (1975)

Notes:

Biochemical oxygen demand (BOD) is defined as parts of oxygen consumed per part of compound during degradation.

MLSS = mixed liquor suspended solids

Table B-2. Toxicity of DEA to Freshwater Aquatic Life

Scientific Name	Common Name	Group	Acute or Chronic	Endpoint	Effect	Exposure Duration (days)	Exposure Type	Chemical Analysis	Concentration (mg/L)	Control Type	Reference	Comments
Primary Data												
Hyalella azteca	Amphipod	Invertebrates	Acute	LC50	Mortality	4	Static	M	344	Satisfactory	Vizon (2006)	Comissioned for this report
Oncorhynchus mykiss	Rainbow trout	Fish	Acute	LC50	Mortality	4	Static	M	460	Satisfactory	Vizon (2006)	Comissioned for this report
Pimephales promelas	Fathead minnow	Fish	Acute	LC50	Mortality	4	Flow-through	Measured	4,710	Satisfactory	Geiger et al. (1990)	
Secondary Data												
Scenedesmus quadricauda	Green algae	Alga	Chronic	IC03	Growth	7	Static	Nominal	4.4	Satisfactory	Bringmann and Kuhn (1980)	Lowest valid datapoint
Ceriodaphnia dubia	Water flea	Invertebrate	Chronic	LC50	Mortality	7	Renewal	Nominal	19	Satisfactory	Cowgill and Milazzo (1991)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	48 h	Static	Nominal	29	Satisfactory	Cowgill et al. (1985)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	48 h	Static	Nominal	30	Satisfactory	Cowgill et al. (1985)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	48 h	Static	Nominal	30	Satisfactory	Cowgill et al. (1985)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	48 h	Static	Nominal	31	Satisfactory	Cowgill et al. (1985)	
Ceriodaphnia dubia	Water flea	Invertebrate	Chronic	EC50	Reproduction	7-10	Renewal	Nominal	34	Satisfactory	Cowgill and Milazzo (1991)	
Daphnia magna	Water flea	Invertebrate	Chronic	EC50	Mortality	11	Static	Nominal	35	Satisfactory	Cowgill and Milazzo (1991)	
Ceriodaphnia dubia	Water flea	Invertebrate	Chronic	EC50	Reproduction	7-10	Renewal	Nominal	37	Satisfactory	Cowgill and Milazzo (1991)	
Ceriodaphnia dubia	Water flea	Invertebrate	Chronic	EC50	Reproduction	7-10	Renewal	Nominal	42	Satisfactory	Cowgill and Milazzo (1991)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	55	Satisfactory	LeBlanc (1980)	
Daphnia magna	Water flea	Invertebrate	Chronic	EC50	Reproduction	9-11	Static	Nominal	57	Satisfactory	Cowgill and Milazzo (1991)	
Daphnia magna	Water flea	Invertebrate	Chronic	EC50	Reproduction	9-11	Static	Nominal	66	Satisfactory	Cowgill and Milazzo (1991)	
Daphnia magna	Water flea	Invertebrate	Chronic	EC50	Reproduction	9-11	Static	Nominal	70	Satisfactory	Cowgill and Milazzo (1991)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	48 h	NR	Nominal	73	Concurrent	Warne and Schifko (1999)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	78	Satisfactory	Cowgill et al. (1985)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	78	Satisfactory	Cowgill et al. (1985)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	99	Satisfactory	Cowgill et al. (1985)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	104	Satisfactory	Cowgill et al. (1985)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	109	Satisfactory	Cowgill et al. (1985)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	109	Multiple	Gersich et al. (1986)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	109	Satisfactory	Cowgill et al. (1985)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	110	Satisfactory	Cowgill et al. (1985)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	110	Multiple	Gersich et al. (1986)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	116	Satisfactory	Cowgill et al. (1985)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	119	Satisfactory	Cowgill et al. (1985)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	124	Satisfactory	Cowgill et al. (1985)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	131	Satisfactory	Cowgill et al. (1985)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	131	Multiple	Gersich et al. (1986)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	135	Satisfactory	Cowgill et al. (1985)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	1	Static	Nominal	158	Multiple	Gersich et al. (1986)	
Entosiphon sulcatum	Flagellate euglenoid	Protozoa	Chronic	IC03	Growth	3	Static	Nominal	160	Satisfactory	Bringmann and Kuhn (1980)	
Ceriodaphnia dubia	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	160	Satisfactory	Cowgill and Milazzo (1991)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	1	Static	Nominal	170	Satisfactory	LeBlanc (1980)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	1	Static	Nominal	173	Multiple	Gersich et al. (1986)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	1	Static	Nominal	235	Multiple	Gersich et al. (1986)	
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	306	Satisfactory	Cowgill and Milazzo (1991)	

Table B-2. Toxicity of DEA to Freshwater Aquatic Life

Scientific Name	Common Name	Group	Acute or Chronic	Endpoint	Effect	Exposure Duration (days)	Exposure Type	Chemical Analysis	Concentration (mg/L)	Control Type	Reference	Comments
Gambusia affinis	Western mosquitofish	Fish	Acute	LC50	Mortality	6	Static	Nominal	560	Satisfactory	Wallen et al. (1957)	
Carassius auratus	Goldfish	Fish	Acute	LC50	Mortality	1	Static	Measured	800	Satisfactory	Bridie et al. (1979b)	
Xenopus laevis	Clawed toad	Amphibian	Acute	LC50	Mortality	2	Static	Nominal	1,174	Satisfactory	De Zwart and Slooff (1987)	
Pimephales promelas	Fathead minnow	Fish	Acute	LC50	Mortality	4	Static	Nominal	1,370	Satisfactory	Mayes et al. (1983)	
Gambusia affinis	Western mosquitofish	Fish	Acute	LC50	Mortality	4	Static	Nominal	1,400	Satisfactory	Wallen et al. (1957)	
Pimephales promelas	Fathead minnow	Fish	Acute	LC50	Mortality	4	Static	Nominal	1,480	Satisfactory	Mayes et al. (1983)	
Pimephales promelas	Fathead minnow	Fish	Acute	LC50	Mortality	4	Static	Nominal	1,550	Satisfactory	Mayes et al. (1983)	
Gambusia affinis	Western mosquitofish	Fish	Acute	LC50	Mortality	2	Static	Nominal	1,550	Satisfactory	Wallen et al. (1957)	
Gambusia affinis	Western mosquitofish	Fish	Acute	LC50	Mortality	1	Static	Nominal	1,800	Satisfactory	Wallen et al. (1957)	
Lepomis macrochirus	Bluegill	Fish	Acute	LC50	Mortality	2	Static	Nominal	1,850	Satisfactory	Turnbull et al. (1954)	
Lepomis macrochirus	Bluegill	Fish	Acute	LC50	Mortality	1	Static	Nominal	2,100	Satisfactory	Turnbull et al. (1954)	
Unacceptable Data												
Carassius auratus	Goldfish	Fish	Acute	LC50	Mortality	1	Static	Measured	>5,000	Satisfactory	Bridie et al. (1979b)	No effect at maximum concentration tested
Microcystis aeruginosa	Blue-green algae	Protozoa	Chronic	NV	Growth	8 d	NR	Nominal	16	NR	Bringmann (1975)	Endpoint not specified
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	1	Static	Nominal	180	Indeterminate	Bringmann and Kuhn (1977a)	Insufficient control information
Microcystis aeruginosa	Blue-green algae	Protozoa	NV	LOEC	Growth	NR	Static	Nominal	16	NR	Bringmann and Kuhn (1978a)	Exposure duration not specified
Microcystis aeruginosa	Blue-green algae	Protozoa	NV	LOEC	Growth	NR	Static	Nominal	17	NR	Bringmann and Kuhn (1978a)	Exposure duration not specified
Scenedesmus quadricauda	Green algae	Alga	Chronic	IC50	Growth	8	Static	Nominal	4	Indeterminate	Bringmann and Kuhn (1978b)	Insufficient control information
Scenedesmus quadricauda	Green algae	Alga	Chronic	IC50	Growth	8	Static	Nominal	10	Indeterminate	Bringmann and Kuhn (1978b)	Insufficient control information
Anacystis aeruginosa	Blue-green algae	Protozoa	Chronic	IC50	Growth	8	Static	Nominal	16	Indeterminate	Bringmann and Kuhn (1978b)	Insufficient control information
Anacystis aeruginosa	Blue-green algae	Protozoa	Chronic	IC50	Growth	8	Static	Nominal	17	Indeterminate	Bringmann and Kuhn (1978b)	Insufficient control information
Scenedesmus quadricauda	Green algae	Alga	NV	LOEC	Growth	NR	Static	Nominal	4	NR	Bringmann and Kuhn (1978c)	Exposure duration not specified
Scenedesmus quadricauda	Green algae	Alga	NV	LOEC	Growth	NR	Static	Nominal	10	NR	Bringmann and Kuhn (1978c)	Exposure duration not specified
Entosiphon sulcatum	Flagellate euglenoid	Protozoa	Acute	NV	Growth	72 h	Static	Nominal	157	NR	Bringmann and Kuhn (1978c)	Endpoint not specified
Scenedesmus quadricauda	Green algae	Alga	NV	NV	Growth	NR	NR	NR	4	NR	Bringmann and Kuhn (1979)	Endpoint not specified
Entosiphon sulcatum	Flagellate euglenoid	Protozoa	NV	NV	Growth	NR	NR	NR	160	NR	Bringmann and Kuhn (1979)	Endpoint not specified
Entosiphon sulcatum	Flagellate euglenoid	Protozoa	NV	NV	Growth	NR	NR	NR	160	NR	Bringmann and Kuhn (1981)	Endpoint not specified
Chilomonas paramecium	Cryptomonad	Protozoa	NV	NV	Growth	NR	NR	NR	1,137	NR	Bringmann and Kuhn (1981)	Endpoint not specified
Uronema parduczi	Ciliate	Protozoa	NV	NV	Growth	NR	NR	NR	1,720	NR	Bringmann and Kuhn (1981)	Endpoint not specified
Daphnia magna	Water flea	Invertebrate	Acute	NOEC	Mortality	24 h	NV	NR	68	NR	Bringmann and Kuhn (1982)	No effect at concentration tested
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	24 h	NV	NR	289	NR	Bringmann and Kuhn (1982)	Insufficient control information
Daphnia magna	Water flea	Invertebrate	Acute	EC50	Mortality	24 h	NV	NR	1,000	NR	Bringmann and Kuhn (1982)	Insufficient control information

Table B-2. Toxicity of DEA to Freshwater Aquatic Life

Scientific Name	Common Name	Group	Acute or Chronic	Endpoint	Effect	Exposure Duration (days)	Exposure Type	Chemical Analysis	Concentration (mg/L)	Control Type	Reference	Comments
Chilomonas paramecium	Cryptomonad	Protozoa	Acute	NV	Growth	48 h	NR	Nominal	1,137	Indeterminate	Bringmann et al. (1980)	Endpoint not specified
Daphnia magna	Water flea	Invertebrate	Chronic	NOEC	Reproduction	9-11	Static	Nominal	3	Satisfactory	Cowgill and Milazzo (1991)	No effect at concentration tested
Daphnia magna	Water flea	Invertebrate	Chronic	NOEC	Reproduction	9-11	Static	Nominal	3	Satisfactory	Cowgill and Milazzo (1991)	No effect at concentration tested
Daphnia magna	Water flea	Invertebrate	Chronic	NOEC	Reproduction	9-11	Static	Nominal	3	Satisfactory	Cowgill and Milazzo (1991)	No effect at concentration tested
Ceriodaphnia dubia	Water flea	Invertebrate	Chronic	NOEC	Reproduction	7-10	Renewal	Nominal	12	Satisfactory	Cowgill and Milazzo (1991)	No effect at concentration tested
Ceriodaphnia dubia	Water flea	Invertebrate	Chronic	NOEC	Reproduction	7-10	Renewal	Nominal	19	Satisfactory	Cowgill and Milazzo (1991)	No effect at concentration tested
Ceriodaphnia dubia	Water flea	Invertebrate	Chronic	NOEC	Reproduction	7-10	Renewal	Nominal	19	Satisfactory	Cowgill and Milazzo (1991)	No effect at concentration tested
Ceriodaphnia dubia	Water flea	Invertebrate	Chronic	NOEC	Mortality	7-10	Static	Nominal	<4.2	Satisfactory	Cowgill and Milazzo (1991)	No effect at concentration tested
Daphnia magna	Water flea	Invertebrate	Chronic	NOEC	Mortality	9-11	Static	Nominal	<4.2	Satisfactory	Cowgill and Milazzo (1991)	No effect at concentration tested
Asellus intermedius	Aquatic sowbug	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Daphnia magna	Water flea	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Dugesia tigrina	Turbellarian, flatworm	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Gammarus fasciatus	Scud	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Helisoma trivolvis	Ramshorn snail	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Lumbriculus variegatus	Oligochaete, worm	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Pimephales promelas	Fathead minnow	Fish	Chronic	LC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Asellus intermedius	Aquatic sowbug	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Daphnia magna	Water flea	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Dugesia tigrina	Turbellarian, flatworm	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Gammarus fasciatus	Scud	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Helisoma trivolvis	Ramshorn snail	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Lumbriculus variegatus	Oligochaete, worm	Invertebrate	Chronic	EC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested
Pimephales promelas	Fathead minnow	Fish	Chronic	LC50	Mortality	4	Static	Nominal	>100	Indeterminate	Ewell et al. (1986)	No effect at maximum concentration tested

Table B-2. Toxicity of DEA to Freshwater Aquatic Life

Scientific Name	Common Name	Group	Acute or Chronic	Endpoint	Effect	Exposure Duration (days)	Exposure Type	Chemical Analysis	Concentration (mg/L)	Control Type	Reference	Comments
Semotilus atromaculatus	Creek chub	Fish	Acute	NOEC	Mortality	1	Static	Nominal	80	Indeterminate	Gillette et al. (1952)	No effect at concentration tested
Semotilus atromaculatus	Creek chub	Fish	Acute	LC50	Mortality	1	Static	Nominal	1,129	Indeterminate	Gillette et al. (1952)	Insufficient control information
Leuciscus idus melanotus	Carp	Fish	Acute	NOEC	Mortality	48 h	NV	NR	500	NR	Juhnke and Luedemann (1978)	No effect at concentration tested
Leuciscus idus melanotus	Carp	Fish	Acute	LC50	Mortality	48 h	NV	NR	1,430	NR	Juhnke and Luedemann (1978)	Insufficient control information
Leuciscus idus melanotus	Carp	Fish	Acute	NOEC	Mortality	48 h	NV	NR	1,530	NR	Juhnke and Luedemann (1978)	No effect at concentration tested
Leuciscus idus melanotus	Carp	Fish	Acute	LC50	Mortality	48 h	NV	NR	1,650	NR	Juhnke and Luedemann (1978)	Insufficient control information
Leuciscus idus melanotus	Carp	Fish	Acute	LC50	Mortality	48 h	NV	NR	1,850	NR	Juhnke and Luedemann (1978)	Insufficient control information
Leuciscus idus melanotus	Carp	Fish	Acute	LC50	Mortality	48 h	NV	NR	2,180	NR	Juhnke and Luedemann (1978)	Insufficient control information
Chlorococcales	Green algae order	Alga	Acute	NV	Growth	24 h	Static	NR	1,000	NR	Krebs (1991)	Endpoint not specified
Chlorococcales	Green algae order	Alga	Acute	NV	Growth	24 h	Static	NR	NV	NR	Krebs (1991)	Endpoint not specified
Daphnia magna	Water flea	Invertebrate	Acute	NV	Mortality	2	Static	Nominal	<24	Satisfactory	LeBlanc (1980)	Endpoint not specified
Daphnia pulex	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	2.2	Indeterminate	Moore et al. (1987)	No relevant DEA data
Daphnia pulex	Water flea	Invertebrate	Acute	EC50	Mortality	2	Static	Nominal	2.6	Indeterminate	Moore et al. (1987)	No relevant DEA data
Gambusia affinis	Western mosquitofish	Fish	Chronic	NV	Mortality	4	Static	Nominal	320	Indeterminate	Wallen et al. (1957)	Endpoint not specified

Notes:

NV = not verified or no value

NR= not recorded

Table B-3. Toxicity of DEA to Marine Aquatic Life

Scientific Name	Common Name	Group	Study Type	Endpoint	Effect	Exposure Duration (days)	Concentration (mg/L)	Exposure Type	Chemical Analysis	Control Type	Reference
Unacceptable Data											
Asterias forbesii	Common starfish	Invertebrate	NV	NR	Population	NR	4	Static	Nominal	Indeterminate	Bringmann and Kuhn (1977b)
Asterias forbesii	Common starfish	Invertebrate	NV	NR	Population	NR	10	Static	Nominal	Indeterminate	Bringmann and Kuhn (1977b)
Skeletonema costatum	Diatom	Protozoa	Chronic	EC50	Physiology	4	103	NR	Nominal	Indeterminate	US EPA (1978)
Americamysis bahia	Opossum shrimp	Invertebrate	Chronic	LC50	Mortality	4	207	NR	Nominal	Indeterminate	US EPA (1978)
Skeletonema costatum	Diatom	Protozoa	Chronic	NOEC	Population	5	216	Static	Nominal	Indeterminate	Cowgill et al. (1989)
Skeletonema costatum	Diatom	Protozoa	Chronic	NOEC	Population	5	216	Static	Nominal	Indeterminate	Cowgill et al. (1989)
Skeletonema costatum	Diatom	Protozoa	Chronic	EC50	Population	5	523	Static	Nominal	Indeterminate	Cowgill et al. (1989)
Cyprinodon variegatus	Sheepshead minnow	Fish	Chronic	NOEC	Mortality	4	540	Static	Nominal	Satisfactory	Heitmuller et al. (1981)
Skeletonema costatum	Diatom	Protozoa	Chronic	EC50	Population	5	548	Static	Nominal	Indeterminate	Cowgill et al. (1989)
Artemia salina	Brine shrimp	Invertebrate	Acute	LC50	Mortality	1	2,800	Static	Nominal	Indeterminate	Price et al. (1974)
Cyprinodon variegatus	Sheepshead minnow	Fish	Acute	LC50	Mortality	1	>540	Static	Nominal	Satisfactory	Heitmuller et al. (1981)
Cyprinodon variegatus	Sheepshead minnow	Fish	Acute	LC50	Mortality	2	>540	Static	Nominal	Satisfactory	Heitmuller et al. (1981)
Cyprinodon variegatus	Sheepshead minnow	Fish	Acute	LC50	Mortality	3	>540	Static	Nominal	Satisfactory	Heitmuller et al. (1981)
Cyprinodon variegatus	Sheepshead minnow	Fish	Chronic	LC50	Mortality	4	>540	Static	Nominal	Satisfactory	Heitmuller et al. (1981)

Notes:

NR = not reported.

NV = not reported in the abstrast and not verified in this literature search.

Table B-4. Toxicity of DEA to Terrestrial Plants

Scientific Name	Common Name	Effect Measurement	Concentration	Endpoint/ Response	Response Site	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
						days				
Medicago sativa	Alfalfa	Length	1,194	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Length	2,109	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Dry Mass	995	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Medicago sativa	Alfalfa	Dry Mass	1,077	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Length	3,194	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Length	4,028	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Dry Mass	2,247	IC25	shoot	14	artificial soil	spiked	Y	Stantec (2006)
Hordeum vulgare	Barley	Dry Mass	858	IC25	root	14	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Length	3,290	IC25	shoot	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Length	3,575	IC25	root	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Dry Mass	1,602	IC25	shoot	21	artificial soil	spiked	Y	Stantec (2006)
Elymus lanceolatus	Northern Wheatgrass	Dry Mass	2,204	IC25	root	21	artificial soil	spiked	Y	Stantec (2006)

Notes:

values presented here are nominal - not corrected for analytical recovery.

Table B-5. Toxicity of DEA to Terrestrial Invertebrates

Scientific Name	Common Name	Effect Measurement	Concentration	Endopint/Response	Test Duration	Media Type	Application Method	Chemical Analysis	Reference
					days				
Eisenia andrei	Earthworm	adult survival	4,141	LC50	35	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	# progeny	171	IC25	63	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	progeny wet mass	2,304	IC25	63	artificial soil	spiked	Y	Stantec (2006)
Eisenia andrei	Earthworm	progeny dry mass	2,136	IC25	63	artificial soil	spiked	Y	Stantec (2006)
Folsomia candida	Springtail	adult survival	8,301	LC50	28	artificial soil	spiked	Y	Stantec (2006)
Folsomia candida	Springtail	# progeny	2,102	IC25	28	artificial soil	spiked	Y	Stantec (2006)

Notes:

values presented here are nominal - not corrected for analytical recovery.

IC25/LC25 values presented where available, otherwise IC50/LC50 presented

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Appendix C - Method for the Extraction of Alkanolamines in Soil by Ionic Reflux Extraction (IRE)

1. Scope

1.1. Applicability

1.1.1 This method is applicable to the measurement alkanolamines in soil.

1.1.2 The results of the test procedure are reported in terms of mg/kg of individual alkanolamine species.

1.1.3 This method has been tested with several different alkanolamine species:

- 1.1.3.1 Monoethanolamine (MEA), CAS# 141-43-5
- 1.1.3.2 Diethanolamine (DEA), CAS# 111-42-2
- 1.1.3.3 Methyldiethanolamine (MDEA), CAS# 105-59-9
- 1.1.3.4 Diisopropanolamine (DIPA), CAS# 110-97-4

1.1.4 The method detection limits (MDL) range from 1 to 20 mg/kg on a dry soil basis. Detection limits will vary based on the alkanolamine species and the analytical technique employed following the extraction process.

1.2 Interferences

1.2.1 Interferences for the detection and quantitation of alkanolamines include any species with similar chromatographic retention time as the target alkanolamines.

1.2.2 Some interferences can be removed by pre-treating (cleaning) the soil extract with hexane.

2. Terminology

2.1. Ionic Reflux Extraction

A preparative technique in which acidified water is allowed to boil and recondense. The acidic condensate flows through the solid sample and is returned to the heated glassware on a continual basis.

3. Summary of Test Method

3.1. Sorption Processes

Alkanolamines are bound to soil particles through several mechanisms. The three principle modes of sorption are *hydrophobic partitioning* of the neutral amine into organic material, *chemical bonding* with active surface groups and *cation exchange* of the positively charged amine with the negatively charged sites on the soil particles. Data collected during the literature search[1] and method development process identified cation exchange as the dominant mode of sorption limiting analytical recovery of alkanolamines spiked into soil samples

3.2. Analytical Challenge

Laboratory investigations have found alkanolamine recovery from soil samples to be generally poor and non-reproducible. Some preliminary testing (unpublished) found recoveries ranging from 10% to 60%, depending on soil type and alkanolamine species. These results were obtained using a 10:1 water extraction and mechanical alkanolamine recoveries. Organic solvent modifiers such as methanol and acetonitrile did not improve recoveries.

3.3. Scientific Rationale and Approach:

The poor recovery in high clay soil samples suggested that cation exchange was the dominant sorption process. Strategies to overcome the cation exchange involved elevated temperature and solvents capable of neutralizing the charged sites on the clay particles. Elevated temperatures and the use of dilute $CaCl_2$ or HCl as extraction solvents produced improved recoveries (30 to 70% in moderate clay soils). Nonetheless, the liquid/solid extraction process seemed to reach an equilibrium point where further recovery of the alkanolamines could not occur. A reflux approach physically separates the bulk solvent from the soil and takes advantage of the equilibrium developed with fresh reflux solvent.

3.4. Selection of extraction solvent:

Both $CaCl_2$ and HCl showed comparable benefits as a solvent, however HCl was chosen as a reflux solvent because of its ability to partition into the vapour phase. The acidic vapour environment improves the recovery of alkanolamines from soil.

3.5 Apparatus

3.5.1 Soxhlet Extraction Glassware or Dean Stark Extraction Glassware.
3.5.2 Standard laboratory glassware, Pipettes and Syringes
3.5.3 Cellulose extraction thimbles
3.5.4 Analytical balance

3.6 Reagents and Materials

3.6.1 All chemicals used for the preparation of reagents and standards are ACS grade or better unless otherwise stated.
3.6.2 Minimum Alkanolamine purity for standard preparation is 98%.

3.7 Safety

This method does not purport to address all of the safety considerations associated with its use. It is the responsibility of the user to establish appropriate health and safety practices.

3.8 Sample Handling, Preservation and Holding Times

Matrix	Sample Container	Minimum Volume	Holding Time (days)	Storage Conditions	Preservation
Soil	Glass or Plastic	100 grams	TBD	<10 ºC	None
Soil Extracts	Glass or HDPE	N/A	TBD	4 ºC	None

4. Procedure

4.1. Sample Preparation

4.1.1 Soil Extraction

4.1.1.1 Using a spatula, mix the sample as well as possible.

4.1.1.2 Accurately weigh out approximately 10 grams of soil, avoiding twigs, large stones and any other non-representative material.

4.1.1.3. Place the 10g sub-sample into a clean cellulose thimble

4.1.1.4. Place the thimble in clean Dean Stark or Soxhlet extraction glassware

4.1.1.5. Add 100mL of 0.01N HCl and reflux for 1 hour. Begin the 1-hour duration from the time the solvent begins to boil.

4.1.1.6. Allow the extract to cool to a safe temperature before transferring to a suitable glass or HDPE extract vial.

4.1.1.7. Filter a suitable portion, approximately 10mL, of the extract is into a glass or HDPE test tube using a 0.45um syringe filter. This process will remove fine clay particles and may remove some color from the extracts.

4.1.1.8. Add 1 mL of hexane to the test tube and mix with a vortex mixer for approximately 10 seconds. Allow to separate. This hexane clean-up step helps to remove organic material that can interfere with the chromatographic measurement of alkanolamines, particularly MEA.

4.1.1.9. Using a disposable pipette or syringe, remove enough of the lower aqueous layer for the analytical determination step.

4.1.2 Matrix Spike

4.1.2.1. Prepare a suitable stock spiking solution containing each of the alkanolamines being tested. The spiking level should fall near the mid level of the analytical calibration range.

4.1.2.2. Spike a 10 gram sample of a randomly selected soil sample with a maximum of 0.5mL of the stock alkanolamine solution. Spike volumes greater than 0.5mL (5%) may alter the soil moisture content and influence the sorption of alkanolamines.

4.1.2.3. Prepare one per batch of 20 samples or less.

4.1.3. Duplicates

4.1.3.1. Prepare a duplicate using a sample chosen at random.
4.1.3.2. Prepare one per batch of 20 samples or less.

4.1.4 Method Blank

4.1.4.1 Accurately weigh 8 -10 g of clean sand into a clean cellulose thimble and carry through the entire analytical process
4.1.4.2 Prepare one per batch of 20 samples or less

4.1.5 Calibration

4.1.5.1 This method describes the extraction process only. Calibration procedures should consistent with the laboratory analytical method employed for the determination of Alkanolamines in Water
4.1.5.2 Suitable analytical techniques include reverse phase liquid chromatography or gas chromatography.

5. Quality Control Requirements

5.1. Method Blank

5.1.1. A method blank is used to ensure there is no systematic contamination throughout the preparation and analysis procedure. The results for the blank must remain below the detection limit.

5.1.1.1. If positive blanks are detected at levels > 10% of sample values, the impacted samples must be re-extracted. Do not subtract blank.

5.2. Matrix Spike

5.2.1. A matrix spike is prepared using a sample from the set to be analyzed. The matrix spike is used to test the effectiveness of the extraction and measurement process. Acceptance limits to be determined.

5.3. Duplicates

5.3.1. The acceptable Relative Percent Difference (RPD) of the duplicate samples is 30% or less for values greater than 5 x the reporting limit (RDL).

6. Calculations and reporting

6.1. Results are quantitated using the external standard method. Alkanolamine content is reported as mg/kg. Data is typically reported on a dry basis: The report must indicate the basis of reporting.

$$[\text{Amine}]\ (\text{mg/kg}) = \frac{A \times D \times V}{W}$$

A = Chromatograph reading (mg/L)
D = Dilution factor (if any)
V = Volume of extract (mL)
W (dry weight) = wet sample weight (g) × (1 – moisture)
(express moisture as a decimal, e.g. 8.5% = 0.085)

7. Detection Limits

7.1. The overall method detection limit is a function of the analytical method selected for the final quantitative analysis. The analytical method used during the development of this extraction method was Liquid chromatography coupled with pulsed amperometric detection. The details of the analytical method can be found the method performance attachment included with this method.

7.2. The extraction method described in this document can be combined with analytical methods capable of measuring alkanolamine species at levels above 0.1 ppm. Using the extraction ratios described in this document this would yield an overall method detection limit of 1.5 ppm in soil.

7.3. There is a detection limit exception for monoethanolamine (MEA) when using the extraction procedure described in this document. The elevated temperatures and acidic conditions can release soil materials that interfere with the chromatographic analysis of MEA. Therefore, overall detection limits for MEA are anticipated to be approximately 5 to 10x higher than the other alkanolamine species.

8. Precision and Bias

8.1. Recovery Bias

The average recovery of alkanolamines across a 3 x 3 x 4 matrix of 3 soil types, 3 spiking levels and 4 alkanolamine species was approximately 97%. This suggests that the extraction technique is suitable for quantitative analysis. Recovery bias is somewhat dependant on soil type. The lowest recoveries occurred for MEA in the high organic carbon (HOC) soil, a very difficult matrix, averaging 60%.

8.2. Extraction Precision

The average RSD of the alkanolamine results across a 2x3x4 matrix of 3 soil types, the mid and high spiking levels and 4 alkanolamine species was approximately 15%. (Both spike values > 5x the RDL). This is typical of between run variability for a variety of organic analyses of soils and suggests that the method is suitable for quantitative analysis. For comparison, the RSD of the 2008 CAEAL petroleum Hydrocarbon (PHC) Performance Test (PT) samples for F2 – F4 (3 fractions x 4 samples x multiple labs) was 20%. Nonetheless, if overall measurement uncertainty for a single sample measurement is insufficient for applications of the data then replicate analysis of samples may be required.